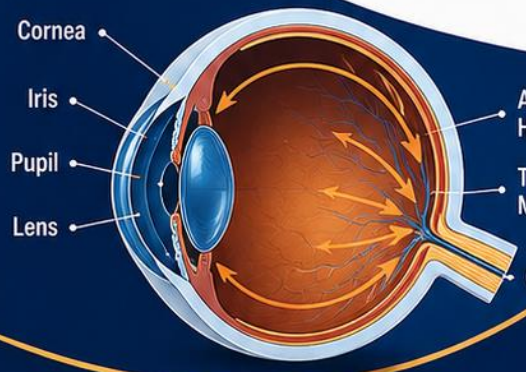
Postgraduate Notes

Glaucoma

Exam Notes



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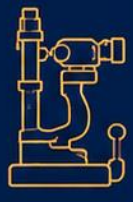

Postgraduate Ophthalmology Exam Notes

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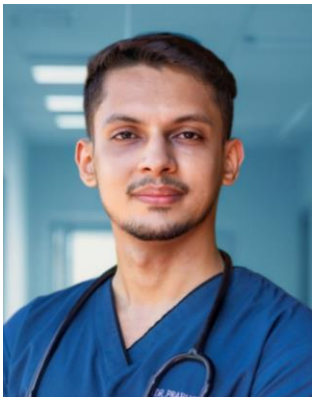

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This book is intended solely as an educational and revision resource for postgraduate ophthalmology trainees and practitioners. While every effort has been made to ensure the accuracy and reliability of the information presented, the author does not guarantee that all content is free from errors or omissions. Readers are encouraged to consult standard textbooks, peer-reviewed literature, institutional guidelines, and current evidence-based recommendations before making clinical decisions.

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Preface

This book is a compilation of notes gathered during my preparation for the Final MD Examination in Ophthalmology. The material has been collected from standard textbooks, lecture notes, journals, online resources, and personal study notes.

Topics have been organized in a chapter-wise format to facilitate quick revision and easy reference. Although there are many excellent ophthalmology resources available, I felt it worthwhile to compile and share these notes with future ophthalmology trainees and colleagues.

This work represents a continuous learning process rather than a definitive textbook. Suggestions, corrections, contributions, and constructive feedback are greatly appreciated and will help improve future editions.

I sincerely hope this book serves as a useful companion in your postgraduate ophthalmology journey.

Good Luck and Best Wishes!

For comments, suggestions, or contributions, please contact:

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IOP MEASUREMENT

1. MANOMETRY

Definition:

Direct measurement of intraocular pressure (IOP) using a needle inserted into the anterior chamber or vitreous, connected to a water or mercury manometer.

Uses:

- Mainly experimental.
- Allows continuous recording of IOP and assessment of physiological or pharmacological changes, mostly in **animal studies**.

Disadvantages:

- Not practical for routine human use.
- Requires **general anesthesia**, which can alter IOP.
- Needle insertion disrupts the **blood–aqueous barrier**, causing prostaglandin release and altering IOP.

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2. TONOMETRY

Definition:

Indirect measurement of IOP using instruments called **tonometers**.

History:

- First by **Donders** (~19th century).
- Mechanical tonometer by **Schiotz** (early 1900s).

Principle:

- Measures IOP by correlating **force applied** to the **deformation of the globe**.

Types:

1. **Indentation (Impression) Tonometry** – measures indentation of the cornea.
2. **Applanation Tonometry** – measures force required to flatten a fixed corneal area.

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❖ **INDENTATION TONOMETRY** *(Schiotz, Herrington, Grants, Maurice)*

Schiotz Tonometry

Principle:

- A plunger indents the cornea; **softer eyes → more indentation**, **harder eyes → less**.
- Measures artificially raised IOP (Pt), converted to baseline IOP (P0) using **Friedenwald tables**.

Design / Components:

- Handle for vertical positioning.
- Footplate rests on cornea.
- Plunger slides freely within shaft.
- Bent lever: short arm on plunger, long arm acts as pointer.
- Weights: 5.5 g (standard); optional 7.5, 10, 15 g.

Technique:

1. Topical anesthesia (2–4% xylocaine).
2. Patient supine, gaze fixed upward.
3. Examiner separates lids, places footplate vertically.
4. Record reading after needle stabilizes.
5. Start with 5.5 g; add weights if scale reading <3.
6. Convert Pt → P0 using **Friedenwald table.**

Sources of Error / Limitations:

- Instrumental: weight differences, friction, footplate curvature.
- Ocular factors: extraocular muscle contraction, accommodation.
- **Ocular rigidity:** high rigidity → falsely high IOP; low rigidity → falsely low.
- Corneal factors: thickness, curvature, scars, astigmatism.
- **Moses effect:** plunger lifted by cornea at low readings.
- Blood volume changes; repeated measurements → lowered IOP.

Advantages:

- Simple, inexpensive, historically widely used.

Sterilization:

- Alcohol cleaning of footplate and barrel; dry before reassembly.

Electronic Schiotz:

- Provides continuous IOP recording; mainly experimental.

Approximate Error:

- ±2 mmHg (normal), ±4 mmHg (high IOP).

❖ **VARIABLE-AREA (FIXED FORCE) APPLANATION TONOMETRY**

Principle: Measures area of cornea flattened by a **known force**.

Examples: Maklakov tonometer, Applanometer, Tonomat, Halberg tonometer, Barraquer tonometer, Ocular Tension Indicator, Glaucotest.

Uses: Screening; less common in modern clinical practice.

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❖ **VARIABLE-FORCE (FIXED AREA) APPLANATION TONOMETRY** *(Goldmann, Perkins, Draeger, Mackay-Marg)*

GOLDMANN APPLANATION TONOMETER (GAT)

Principle:

- Based on **modified Imbert–Fick law:** P = F / A.
- The pressure inside an ideal, dry, thin-walled, perfectly spherical object is equal to the force required to flatten (applanate) its surface divided by the area flattened.
- In applanation tonometry, the force to flatten a 3.06 mm corneal area equals the IOP, as corneal rigidity and surface tension cancel, making measured pressure approximate true IOP.

Design / Components:

- Mounted on slit-lamp.
- Double biprism tip for applanation.

Technique:

1. Topical anesthetic + fluorescein.(0.25%)
2. Patient seated at slit-lamp, forehead on headrest, gaze straight. (60° angulation)
3. Prism centered on corneal apex; cobalt blue filter on.
4. Adjust force until **inner edges of semicircular mires touch**.
5. Dial reading ×10 → IOP (mmHg).

Sources of Error:

- Fluorescein thickness: too thick → overestimate; too thin → underestimate.
- Eyelid pressure, globe squeezing → falsely high.
- **CCT:** Thin → underestimate; thick → overestimate.
- Astigmatism >3D → rotate prism 90° and average readings. (WTR: underestimates, ATR: overestimates)
- Corneal edema → falsely low IOP.
- Calibration errors, pulse pressure, repeated readings → minor errors.
- Corneal Irregularity

CCT Correction:

- ~0.7 mmHg per 10 µm deviation from 520 µm (Dredsner table).

Sterilization:

- Soak in diluted Na-hypochlorite (1:10), H_2O_2 3%, or isopropyl alcohol 70%; disposable prisms reduce infection risk.

Hand-held Goldmann-type tonometers:

- Perkins, Draeger; portable, same principle.

❖ **OTHER TONOMETERS**

Newer Tonometers: Dynamic Contour Tonometer (Pascal) ,Ocular Response Analyzer (ORA), Rebound Tonometer, Proview Phosphene Tonometer

a) Non-Contact Tonometer (NCT / Air-Puff)

Principle: Applanation by air pulse; measures time/force required to flatten cornea.

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Design: Automatic air-puff; digital readout; no contact.
Technique: Patient seated; air pulse delivered; corneal deformation detected optically. Average ≥3 readings.
Advantages: Non-invasive, no anesthesia, safe for mass screening.
Disadvantage: Tear film damage, False Negative & positive
Limitations: Startle response → error; less accurate than GAT; affected by CCT or irregular cornea.

b) Dynamic Contour Tonometry (Pascal)

Principle: Contour-matched applanation; IOP independent of corneal properties.
Design: Tip with pressure sensor; mounted on slit-lamp; digital output.
Technique: Tip gently contacts cornea; sensor detects corneal pulse; digital IOP + ocular pulse amplitude.
Advantages: Less affected by CCT; measures physiological IOP, No fluorescein use, No mechanical calibration
Limitations: Expensive, mainly research/specialized clinics.

c) Rebound Tonometry (iCare)

Principle: Probe rebounds off cornea; deceleration and contact time correlate with IOP.
Design: Hand-held; solenoid propels probe; sensing coil; disposable tips.
Technique: No anesthesia; multiple readings (4–10); digital output.
Advantages: Portable; pediatric and screening use; safe; disposable tips.
Limitations: Slight overestimation vs GAT; sensitive to movement; less accurate in high IOP or irregular corneas.

d) Tono-Pen

Principle: Electronic applanation; flattens small corneal area, converts force to IOP.
Design: Hand-held, strain gauge sensor, digital display, disposable tip.
Technique: Topical anesthesia; perpendicular to cornea; multiple readings averaged.
Advantages: Best For Corneal Scar, Portable, rapid, suitable for children, supine, ICU, irregular corneas.
Limitations: Slight overestimation at high IOP, sensitive to movement, less precise than GAT.
Use: Pediatric glaucoma, ICU/bedside, screening.

e) Other Special Tonometers

- **Transpalpebral / Diastolic tonometer:** IOP through eyelid; no corneal contact.

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- **Ocular Response Analyzer (Reichert):** Measures IOP + corneal hysteresis; useful post-LASIK.
- **SmartLens / Implantable sensors:** Continuous IOP monitoring; experimental.
- **Proview-Pressure Phosphene:** Patient self-monitoring via entopic phenomenon.

Exam Notes

- GAT is the gold standard tonometer because it is more accurate, highly reproducible, less affected by scleral/ocular rigidity and axial length, and is ideal for serial follow-up of glaucoma patients.
- GAT applanates a corneal area of 7.35 mm^2 (3.06 mm diameter), where tear film surface tension and corneal rigidity neutralize each other, enabling accurate IOP measurement.
- Volume of fluorescein dye dispensed by GAT: 0.5 µL.
- Knob is kept at 1 (10 mmHg), not 0, because at 0, micro-vibrations of the prism may cause corneal epithelial abrasions/erosions.
- Calibration: 0.1 g force = 1 mmHg IOP.
- GAT prism can potentially transmit adenovirus (most common), HSV, HBV, and theoretically HIV if not adequately disinfected.

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Factors Affecting Intraocular Pressure (IOP)

A. Local Factors

- Rate of aqueous humor formation
- Resistance to aqueous outflow *(most important)*
- Episcleral venous pressure (EVP)
- Pupillary dilatation
- Refractive error: Myopia → ↑ IOP

Drugs affecting IOP

- Steroids → ↑ IOP
- Alcohol → ↓ IOP *(↓ aqueous production)*
- Cannabis → ↓ IOP *(temporary)*

B. General Factors

- Heredity
- Age: ↑ after 40 years
- Female sex: Slightly ↑ risk
- Diurnal variation: Morning ↑, Evening ↓
- Posture: Supine ↑
- Seasonal variation: Winter ↑
- Blood pressure: Mild ↑ BP → Mild ↑ IOP
- Hormonal stimulation: ↑ IOP
- Exercise: ↓ IOP
- Stress: ↑ IOP

Factors that Increase IOP (↑ IOP)

↑ Episcleral Venous Pressure (EVP) due to:

- Valsalva maneuver
- Breath holding
- Playing wind instruments
- Tight collar/tie
- Bending over
- Supine position
- External pressure on the eye
- Lid squeezing
- Blepharospasm

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Target IOP

Target IOP is the **IOP level below which the risk of glaucoma progression is minimal**, thereby preventing further structural (optic disc) and functional (visual field) damage.

Target IOP (TP) = Initial IOP (IP) × [1 – (IP/100)] – Z ± 2
Where:

- **IP** = Initial (baseline) IOP
- **Z** = Functional status (optic disc damage/visual field loss)

Z Score	Clinical Status
0	Glaucoma suspect (Low risk)
1	Glaucoma suspect (High risk)
2	Early glaucoma
3	Moderate glaucoma
4	Severe glaucoma
5	End-stage glaucoma

AAO Target IOP Recommendations

Condition	Target IOP Reduction
Ocular Hypertension (OHT)	↓ 20%
Mild (Early) Glaucoma	↓ 20%
Moderate Glaucoma	↓ 30%
Advanced/Severe Glaucoma	↓ 35–40%
Normal Tension Glaucoma (NTG)	↓ 30%

AAO Grading of Glaucoma Severity

Severity	Optic Disc Changes	Visual Field Defect (VFD)
Mild (Early)	Present	No visual field defect
Moderate	Present	Visual field defect in one hemifield, not within 5° of fixation
Severe (Advanced)	Present	Visual field defect in both hemifields, or within 5° of fixation

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PHASING

IOP Phasing = Serial measurement of intraocular pressure at fixed intervals (usually every 2 hours) over 8–24 hours to assess **diurnal variation** and detect **peak IOP spikes**.

Also called:

- Diurnal IOP curve
- Day phasing
- 24-hour IOP monitoring (if extended)

Rate of aqueous production is low during sleep & increases during day in response to circulating catecholamines.

PHYSIOLOGICAL BASIS

🔷 Aqueous Humour Dynamics

Produced by ciliary body → flows through pupil → anterior chamber → drains via:

- Trabecular meshwork → Schlemm's canal (conventional pathway)
- Uveoscleral pathway (unconventional pathway)

Normal IOP: **10–21 mmHg** (some texts: up to 24 mmHg)

IOP = Balance between aqueous production and outflow.

DIURNAL VARIATION OF IOP

- Normal fluctuation: **2–6 mmHg**
- In glaucoma: may exceed **8–10 mmHg**
- Peak commonly in **early morning**
- Lowest typically in afternoon/evening
- Some patients show **nocturnal peaks**

Important Concept

A single office IOP may:

- Miss peak spikes
- Underestimate true disease severity
- Mislead treatment decisions

Diurnal variation d/t circadian rhythm by fluctuation of blood level of adrenocortical steroids &

Postural variation d/t increased EVP while lying down

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INDICATIONS (EXAM FAVORITE)

Absolute Indications

1. Borderline IOP (Ocular Hypertension)
2. Progressive glaucoma with normal clinic IOP
3. Normal Tension Glaucoma (NTG)
4. Suspected IOP spikes
5. To evaluate treatment efficacy

Relative Indications

- Pre-surgical decision making
- Poor control despite multiple medications
- Steroid responders

TYPES OF PHASING

Type	Description
Day Phasing	8am–4pm (every 2 hours)
Extended Phasing	8am–8pm
24-Hour Phasing	Includes nocturnal readings
Home Monitoring	Rebound tonometry (selected cases)

PROCEDURE (CLINICAL PROTOCOL)

Setting

- Usually performed in glaucoma clinic
- Measurements every **2 hours**

Technique

Most commonly:

- **Goldmann Applanation Tonometer**

Steps:

1. Topical anesthetic instilled
2. Fluorescein dye applied
3. Applanation under cobalt blue light
4. Record IOP in mmHg
5. Document time of measurement

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INTERPRETATION OF RESULTS

A. Diurnal Variation

- Normal: ≤ 6 mmHg
- Suspicious: 6–8 mmHg
- Abnormal: > 8 mmHg

B. Peak IOP

- Peak > 21 mmHg significant in OHT suspects
- In NTG, even small spikes (18–20 mmHg) may be relevant

C. Mean IOP

Average of all readings

Important in monitoring progression

D. Pattern Recognition

- Morning peak pattern
- Reverse diurnal pattern
- Flat curve (unlikely glaucoma)

ROLE IN SPECIFIC GLAUCOMA TYPES

🔷 **Primary Open Angle Glaucoma (POAG)**

- Detects hidden IOP spikes
- Explains progression despite "controlled" IOP

🔷 **Normal Tension Glaucoma**

- Detects undiagnosed IOP elevations

🔷 **Ocular Hypertension**

- Helps decide treatment initiation

🔷 **Primary Angle Closure Glaucoma**

- Identifies intermittent angle closure spikes

ADVANCED / MODERN ALTERNATIVES

🔷 **Rebound Tonometry**

- Portable
- No anesthesia required

🔷 **Continuous Monitoring Contact Lens**

Example:

- **Sensimed Triggerfish**
  - 24-hour monitoring
  - Detects circadian fluctuation

CLINICAL PEARLS (EXAM GOLD)

- A single normal IOP does NOT rule out glaucoma.
- Highest IOP reading determines risk.
- Target IOP must account for peak pressure.

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- Large diurnal variation = independent risk factor.
- Phasing is especially valuable in NTG suspects.

TARGET IOP MODIFICATION BASED ON PHASING

If peak IOP:

- Slightly elevated → add single medication
- Markedly elevated → combination therapy
- Persistent spikes → consider laser/surgery

Example interventions:

- Prostaglandin analogues (better 24-hour control)
- SLT (reduces fluctuation)
- Filtering surgery (reduces peak amplitude)

PATIENT COUNSELLING POINTS

- It is a long test (8 hours or more).
- Measurements every 2 hours.
- Mild discomfort from drops.
- Can stay in clinic or leave between readings.
- Bring reading material/companion.

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GONIOSCOPY

Introduction Greek *gōnia* (angle) + *skopein* (to view): Clinical technique to examine the anterior chamber (AC) angle. Essential for glaucoma diagnosis, risk assessment, and therapeutic planning.

- **Direct goniolens:** Koeppe, Barkan, Swan–Jacob, Richardson.
- **Indirect goniolens:** Goldmann, Zeiss, Posner, Sussman, Volk.

Principles: In gonioscopy, the anterior chamber angle cannot be seen directly due to total internal reflection at the cornea–air interface (critical angle ~46°). A goniolens or prism replaces this interface with a cornea–lens interface of similar refractive index, allowing light from the angle to exit and be visualized.

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2. Types

Feature	Direct Gonioscopy	Indirect Gonioscopy
Lens	Steeply curved goniolens	Mirror/prism goniolens,
Patient Position	Supine	Seated at slit-lamp
View	Direct, panoramic	Reflected, inverted
Field of View	Wide	Narrower
Magnification	Observer's eye	Slit-lamp
Dynamic/Indentation	Limited	Possible (four-mirror)
Advantages	Detailed view, surgical use	Faster, better illumination, routine clinic
Limitations	Impractical, no slit-lamp benefit, Expensive	Mirror inversion, pressure artifacts
Best For	Surgery, infants, Time Consuming	Glaucoma assessment, angle closure, Quick

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3. Indications

A. Diagnostic

- Classify glaucoma: **open-angle vs. angle-closure**
- Assess AC angle recess & **risk of closure**
- Identify **plateau iris**
- Detect **neovascularization**
- Evaluate angle **pigmentation** & pseudoexfoliation
- Post-trauma: **angle recession, cyclodialysis, foreign bodies**
- Detect **neoplasms** (ciliary body tumors)
- Evaluate **Schlemm's canal** (blood, EVP)
- Detect **Kayser-Fleischer rings**
- Post-surgical: trabeculectomy, glaucoma implants

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- Congenital anomalies: **aniridia, abnormal iris insertion**

B. Therapeutic

- Laser: trabeculoplasty, iridoplasty, cyclophotocoagulation
- **Goniotomy, gonioplasty, trabectome**
- Reopen trabeculectomy ostia
- Implant insertion into Schlemm's canal
- Indentation for **acute angle-closure**

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4. Techniques

A. Direct Gonioscopy

- Lens on cornea with topical anesthetic + saline
- Examine systematically: **nasal → superior → temporal → inferior**
- Advantages: panoramic, flexible, detailed
- Disadvantages: time-consuming, less slit-lamp clarity

B. Indirect Gonioscopy

- **Coupling agent:** Hypromellose, HPMC
- Lens on cornea at slit-lamp
- Mirror image: **angle opposite mirror**, orientation inverted
- **Methods:**
  - Non-indentation: minimal pressure, standard view
  - Dynamic/manipulative: gaze/lens adjustments ("over-the-hill view")
  - Indentation: differentiate **appositional vs. synechial closure**, push aqueous into angle (Zeiss, Posner, Sussman)

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5. Angle Structures – Identification

Structure	Description / Appearance	Clinical Significance
Schwalbe's line	Termination of Descemet's membrane; anterior landmark	Marks start of angle
Trabecular meshwork	Anterior non-pigmented, posterior pigmented	Functional part, IOP regulation
Scleral spur	Whitish band posterior to TM	Ciliary body attachment
Ciliary body	Pink/brown/grey band posterior to spur	Iris insertion evaluation
Iris processes	Finger-like extensions to scleral spur	Distinguish from PAS
Blood vessels	Radial at base normal; abnormal crossing spur	Detect pathology
Schlemm's canal	May show blood	Physiological/pathological clue

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6. Functional Gonioscopy

- **Gonioscopy in situ:** Evaluates occludable angles without lens distortion
- **Dynamic gonioscopy:** Visualize steep or narrow angles
- **Indentation:** Confirms **appositional vs. synechial closure**, useful in **acute angle-closure**

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7. Clinical Uses

- Differentiate **POAG vs. PACG**
- Diagnose **congenital/secondary glaucomas**
- Detect **tumors, cysts, foreign bodies, Kayser-Fleischer rings**
- Post-surgical evaluation: trabeculectomy, micro-implants
- Acute angle-closure attack management
- Post-trauma: hyphaema, synechiae, angle/cyclodialysis
- Post-vitrectomy secondary glaucoma evaluation

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8. Critical Assessment Points

- **Angle openness:** Visible structures
- **Irido-trabecular contact:** Appositional vs. synechial
- **Evidence of contact:** Pigment clumps
- **Reversibility:** via indentation
- **Angle pigmentation:** Mild/moderate/heavy
- **Iris configuration**
- **New vessels:** NVI
- **Prominent iris processes / anterior Schwalbe's line**
- **PAS vs. iris processes**
- **Trabeculectomy / micro-implant patency**
- **Post-trauma features:** Hyphaema, synechiae, angle recession, cyclodialysis

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9. Limitations & Artefacts

- Contact procedure → discomfort, infection risk
- Not suitable: open-globe, early post-trauma
- Corneal edema/opacities limit view
- Excessive pressure → angle artifacts
- Slit-lamp light → pupil constriction, angle distortion
- Wide **inter-observer variability**
- Artefacts: lens residue, mirror misplacement, corneal folds

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10. Disinfection & Sterilization

- **Cleaning:** Cold water, mild soap
- **Disinfection:** 70% isopropyl alcohol (5–15 min), 3% H_2O_2, povidone-iodine
  - 2% glutaraldehyde – 20 min
  - 10% bleach – 10 min

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- **Sterilization:** Ethylene oxide (56°C, 1h), autoclaving (glass lenses)
- **Avoid:** Formalin, phenol (lens damage)

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11. Pathological Findings

Finding	Associated Condition
Peripheral anterior synechiae	PACG, uveitis, ICE syndrome
Neovascularization	Neovascular glaucoma, chronic uveitis
Hyperpigmentation	Physiological, pigment dispersion, pseudoexfoliation, post-trauma, post-YAG iridotomy
Trauma signs	Angle recession, trabecular dialysis, cyclodialysis, foreign bodies
Blood in Schlemm's canal	Physiological or pathological (Sturge-Weber, carotid-cavernous fistula)

Iris process vs PAS

Iris Process	PAS (Peripheral Anterior Synechiae)
lacy, fenestrated	Solid, non-fenestrated
Underlying angle structures visible through spaces	Occludes view of underlying structures

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Normal BV vs MVA

Normal BV	NVA
Broad	Fine
Do not cross SS	Cross SS
Do not arborize	Branch, arborize

High Yield Notes

- In Corneal edema gonioscopy can be performed after applying anhydrous glycerin to clear the cornea temporarily.
- Cycloscopy (Cyclodialysis cleft evaluation): Direct visualization of the ciliary processes, useful in aniridia and after peripheral iridectomy (PI)/cyclodialysis evaluation.

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Sampaolesi Line vs Pigmented Trabecular Meshwork (PTM)

Sampaolesi Line	PTM (Pigmented Trabecular Meshwork)
Anterior to corneal wedge (anterior to Schwalbe's line)	Posterior to corneal wedge
Dark, coarse, irregular	Fine, uniform brown
Salt & pepper	Brown sugar
Discontinuous	Continuous
Abnormal	Normal
Indicates excess pigment deposition	Normal angle landmark

Causes of Increased Angle Pigmentation

- Pseudoexfoliation syndrome (PXF/PXS)
- Pigment Dispersion Syndrome (PDS)/Pigmentary glaucoma
- Previous ocular trauma
- Post-laser Peripheral Iridotomy (PI)
- Aphakia
- Pseudophakia

Causes of Blood in Schlemm's Canal

- Raised episcleral venous pressure (EVP) due to:
  - Carotid–cavernous fistula (CCF)
  - Sturge–Weber syndrome (SWS)
  - Superior vena cava (SVC) obstruction
  - Dural arteriovenous shunt (DAVF)
- After gonioscopy (transient reflux)
- Ocular hypotony
- Post-trabeculectomy hypotony

- **Goldmann 3-Mirror Lens**

Mirror	Angle	Area Visualized
Equatorial mirror	73°	Posterior pole → Equator
Peripheral retinal mirror	67°	Equator → Ora serrata
Gonioscopy mirror	59°	Anterior chamber angle

- **Goldmann single-mirror lens (62°)** is used only for gonioscopy.
- **Zeiss 4-mirror lens (64°)** permits indentation (dynamic/compression) gonioscopy without requiring coupling fluid.

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Optic Nerve Head (ONH) Changes in Glaucoma

1. Papillary Changes (Cup Changes)

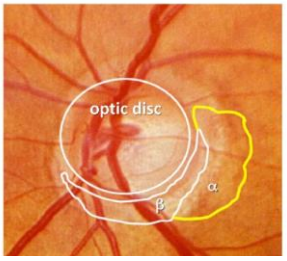
- Vertical enlargement of cup *(earliest disc change)*
- Cup-disc asymmetry >0.2
- Focal neuroretinal rim notching
- Concentric enlargement of cup
- Deepening of cup
- Saucerization → Diffuse neuroretinal rim (NRR) thinning

2. Peripapillary Changes

- Retinal nerve fiber layer (RNFL) defects *(often one of the earliest structural signs)*
- Peripapillary atrophy (PPA) (especially β-zone)
- Drance (splinter) disc hemorrhage → Marker of glaucoma progression

3. Vascular Changes

- Baring of circumlinear vessels
- Bayonetting sign
- Collateral vessel formation
- Nasal shifting of retinal vessels
- Attenuation (narrowing) of vessels



Peripapillary Atrophy	**α-Zone**	**β-Zone**
Location	Peripheral to β-zone	Immediately adjacent to optic disc (usually temporal)
Appearance	Irregular hypo-/hyperpigmentation	Well-defined pale white area
Retinal Pigment Epithelium (RPE)	Irregular pigmentation	Complete loss of RPE
Photoreceptors	Intact	Lost
Sclera & Choroidal Vessels	Not visible	Visible (bare sclera with large choroidal vessels)
Seen in	Normal aging, myopia, glaucoma	Glaucoma (classical finding)
Clinical Significance	Nonspecific	Strong marker of glaucomatous optic neuropathy

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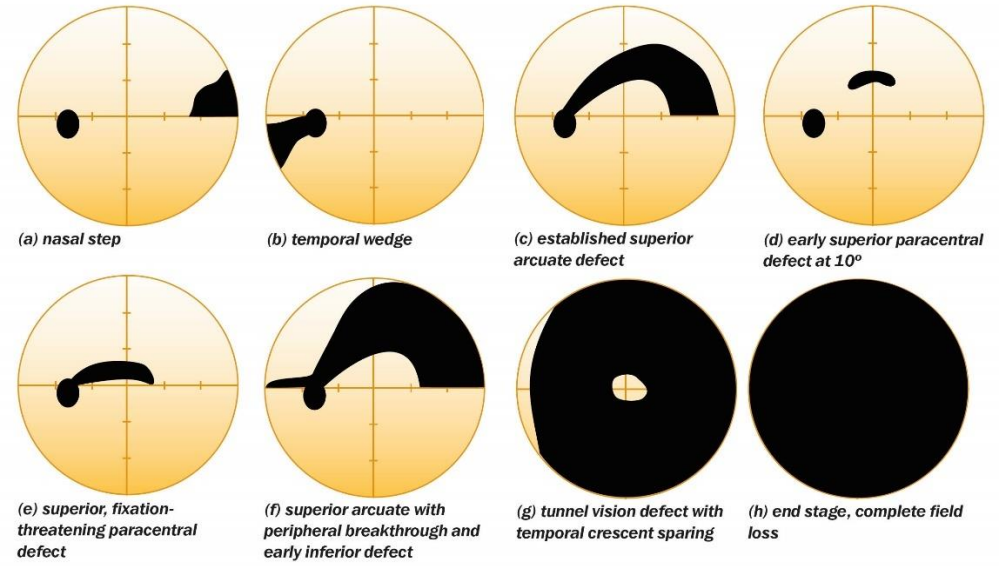
Glaucomatous Visual Field Defects (VFD)

1. **Isopter constriction** → *Earliest visual field defect*
2. **Baring of blind spot**
3. **Paracentral scotoma** → *Earliest clinically significant VFD*
4. **Seidel's scotoma** (Sickle-shaped extension of blind spot)
5. **Arcuate (Bjerrum) scotoma**
6. **Ring (Double arcuate) scotoma**
7. **Roenne's nasal step** (Temporal wedge defect)
8. **Peripheral visual field loss**
9. **Advanced glaucomatous field defect** → Only small central/temporal island of vision remains ("Tunnel vision")


(a) nasal step
(b) temporal wedge
(c) established superior arcuate defect
(d) early superior paracentral defect at 10°
(e) superior, fixation-threatening paracentral defect
(f) superior arcuate with peripheral breakthrough and early inferior defect
(g) tunnel vision defect with temporal crescent sparing
(h) end stage, complete field loss

High-Yield Exam Notes

- **Increased cupping:** Backward bowing of the lamina cribrosa due to loss of retinal ganglion cell axons in the neuroretinal rim.
- **Bean-pot cupping**: Extreme posterior displacement of the lamina cribrosa with undermining of the disc margin (advanced glaucoma).
- **RNFL defect types**: Localized, wedge-shaped, slit-shaped, diffuse, and mixed.
- **Superior & inferior poles**: Larger laminar pores and less glial/connective tissue support → earliest glaucomatous damage.
- **Central visual field preserved**: Thick, densely packed central laminar septae protect papillomacular fibers until late glaucoma.
- **Neurological visual field defect (glaucoma mimic):** Respects the vertical meridian, often has RAPD and asymmetric dyschromatopsia, and contralateral RAPD may be absent/normal depending on the lesion.

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PRIMARY OPEN-ANGLE GLAUCOMA

1. Definitions

Primary open-angle glaucoma (POAG) is a chronic, progressive, multifactorial optic neuropathy characterized by:

- Open, normal-appearing anterior chamber angle
- Optic nerve head and RNFL damage with corresponding visual field defects
- No secondary cause for the glaucoma
- Elevated IOP may be present, but is considered a major modifiable risk factor, not a diagnostic requirement

2. Subtypes:

- **Classic POAG**: Elevated IOP + ONH/VF changes
- **Normal-Tension Glaucoma (NTG)**: ONH/VF damage with IOP ≤21 mmHg
- **Ocular Hypertension (OHT)**: Raised IOP without structural/functional glaucomatous damage

POAG Suspect: A patient with open angles on gonioscopy and no secondary cause for glaucoma, plus ≥1 of the following risk factors in either eye:

- IOP > 21 mmHg (consistent, applanation)
- Optic nerve head changes suggestive of glaucoma (rim thinning/sloping or disc hemorrhage)
- Disc asymmetry: CDR difference > 0.2
- RNFL/Ganglion cell loss (localized or diffuse) without another cause
- Suspicious early visual field defects
- Genetic mutation increasing COAG risk

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3. Epidemiology

- POAG prevalence: ~2% in adults >40 yrs; ~10% in >80 yrs
- OHT prevalence: 4–10% in adults >40 yrs
- Higher prevalence: African, Hispanic, Asian populations
- Faster progression: African ancestry, exfoliation glaucoma, older age, women

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4. Risk Factors

Major (Strongly Associated)

- Elevated IOP (modifiable)
- Age >40 yrs; >60 yrs → 7× higher risk
- Race: African/Latino ancestry → earlier, severe disease
- Family history / Genetics: siblings 10×, offspring 4×; genes: MYOC, OPTN, WDR36
- Thin central corneal thickness (<555 µm) → 3× higher risk

Other / Moderate

- Diabetes mellitus, cardiovascular factors, thyroid disorders
- Optic nerve head features: large discs, high cup:disc ratio
- Myopia, migraine (esp. NTG), sleep apnea

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5. Pathophysiology

A. IOP Elevation (Mechanical / TM Dysfunction)

↑ IOP → Excess glutamate → NMDA receptor activation → ↑ Ca^{2+} influx → NO pathway activation → RGC apoptosis.

- **Site of resistance**: Juxtacanalicular trabecular meshwork (TM) + Schlemm's canal
- **Mechanisms**:
  - Accumulation of extracellular material (GAGs, pigment, deposits)
  - TM endothelial cell loss, giant vacuole reduction → decreased outflow
  - TM sclerosis, narrowed spaces, Schlemm canal collapse, collector channel narrowing
- **Theories**: Aging, oxidative stress, immunogenic mechanisms, altered corticosteroid metabolism, TGF-β toxicity

B. Optic Neuropathy

- Primary: RGC death → optic nerve atrophy → VF defects
- **Mechanisms**:
  - Mechanical Theory (IOP dependent): Lamina cribrosa deformation, impaired axoplasmic flow, translaminar pressure differences – *direct compression & axonal death*
  - Vascular Theory (IOP Independent): Impaired autoregulation, nocturnal hypotension, vasospasm – *impaired perfusion of ON*
  - Secondary: Excitotoxicity (glutamate, NO, ROS)
- Multifactorial: Genetics + mechanical stress + vascular compromise + neurotoxicity + impaired repair

C. Molecular / Cellular

- Extracellular matrix remodeling
- Autoimmune processes
- Oxidative stress, mitochondrial dysfunction
- Loss of neurotrophic support

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6. Genetics

- Polygenic, complex inheritance
- Known mutations - MYOC (myocilin gene), OPTN (optineurin gene), TBK1, WDR36, NTF4 gene.
- Family history increases risk 3–13×
- Gene–environment interactions important

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7. Clinical Features

Symptoms

- Mostly asymptomatic until significant VF loss

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- Occasional: mild headache/eyeache, halos (acute IOP spikes), delayed dark adaptation, difficulty in near work, scotomas

Signs

Anterior Segment

- Often normal; possible thin CCT (<555 µm)
- Pupils: RAPD if asymmetric, sluggish late
- Cornea: mild haze in some

IOP Patterns

- Early: intermittent elevation, exaggerated diurnal variation
- Late: persistent elevation (30–45 mmHg), usually symmetric
- Diurnal Variation Test: variations >6 mmHg suspicious, >8 mmHg strongly suggestive

Optic Nerve Head

- Early: vertical cup elongation, rim thinning superior/inferior, cup asymmetry >0.2, splinter hemorrhages, nasal vessel shift, lamellar dots
- Advanced: marked cupping (0.7–0.9), rim notching, ISNT rule violation
- End-stage: glaucomatous optic atrophy

Visual Fields

- Initial: paracentral wing-shaped scotoma → Seidel's scotoma → arcuate/Bjerrum → central nasal step → tubular vision
- VF defects appear after ~40% axonal loss
- Always correlate VF with ONH and IOP

---

8. Diagnostic Investigations

- **Tonometry**: Goldmann applanation
- **CCT**: adjust IOP interpretation
- **Diurnal / 24h IOP monitoring** (Sensimed CLS)
- **Gonioscopy**: rules out secondary glaucoma
- **Optic disc documentation / OCT / HRT / GDx**
- **Perimetry**: HFA, automated VF testing
- **Nerve Fiber Layer Analyzer**: early RNFL damage
- **Provocative Tests**: Water Drinking Test, less commonly others

Differential Diagnosis

- OHT, NTG, PACG, pigment dispersion, pseudoexfoliation, steroid-induced glaucoma, Posner-Schlossman syndrome, physiological cupping, myopic VF defects

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9. Screening Recommendations

- Baseline exam at 40 yrs for average-risk adults
- Earlier for high-risk (family history, African ancestry, myopia)
- Sensitive combo: OCT + IOP + ONH exam

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10. Natural History / Progression

- COAG/OHT: IOP 21–25 → ~14.4 yrs to end-stage; IOP 25–30 → ~6.5 yrs; >30 → ~2.9 yrs
- NTG: 1/3 progress at 3 yrs, 1/2 at 7 yrs
- Faster progression: women, older age, disc hemorrhage, migraines
- Exfoliative glaucoma: highest progression (~93%)

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11. Risk Stratification & When to Treat

- **High-risk**: thin cornea, high baseline IOP, large cup-disc ratio, disc hemorrhage → treat
- **Moderate-risk**: treat or observe based on factors
- **Low-risk**: periodic observation
- Goal: IOP reduction ≥20% or <24 mmHg

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12. Management

A. Medical Therapy (First-line)

- Identify target IOP:
  - Mild/moderate damage: 16–18 mmHg
  - Severe damage: 12–14 mmHg
- Start monotherapy → combination if needed
- Classes:
  - Beta-blockers: Timolol, Betaxolol, Levobunolol
  - Prostaglandin analogues: Latanoprost, Travoprost, Bimatoprost
  - Adrenergic agents: Brimonidine, Epinephrine/Dipivefrine
  - Topical CAIs: Dorzolamide, Brinzolamide
  - Miotics: Pilocarpine
  - Oral CAIs: Acetazolamide (short-term)
  - Neuroprotective agents (experimental)

B. Laser Trabeculoplasty

- ALT/DLT: shrink TM collagen, IOP ↓ 8–16 mmHg
- SLT: selective TM cell targeting, repeatable
- Indications: uncontrolled IOP, intolerance, non-compliance
- Complications: transient IOP spike, inflammation, rare hemorrhage/synechiae

C. Surgical Therapy

- Indications: uncontrolled IOP, advanced disease, non-compliance
- Procedures: Trabeculectomy (common), other filtration surgeries, cyclodestructive procedures (refractory)
- Follow-up: stable → 3–4 months; high-risk/progressive → more frequent
- Monitor: IOP, VF, ONH, RNFL, gonioscopy

---

13. Prognosis

- Untreated → irreversible blindness; ~35% reach end-stage in 10 yrs

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- IOP lowering reduces progression (EMGT: 50% reduction over 6 yrs)
- Rate influenced by baseline optic nerve damage, IOP, genetics, compliance, systemic health

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OCULAR HYPERTENSION

Definition

- Ocular Hypertension (OHT) is defined as:
  - Intraocular Pressure (IOP) >21 mmHg
  - Open angles on gonioscopy
  - No structural glaucomatous optic nerve damage
  - No functional visual field loss
- Normal population IOP distribution:
  - Mean IOP: ≈ 16 mmHg
  - Normal range (mean ± 2 SD): 11–21 mmHg
  - Population studies: ***Beaver Dam Eye Study (USA), Baltimore Eye Survey (USA), Rotterdam Study (Europe), Blue Mountain Study***
  - These studies report mean IOP ≈ 15–16 mmHg with SD ≈ 2.5–3 mmHg, yielding the 11–21 mmHg normal range.
  - In individuals >70 years, upper limit may reach 23 mmHg
- Prevalence: 4–7% of population >40 years have IOP >21 mmHg with no glaucomatous damage.

EPIDEMIOLOGY

- Affects **4.5–9.4%** of middle-aged and elderly adults in the US.
- Prevalence varies by ethnicity:
  - Afro-Caribbean (Barbados Eye Study): **12.6%**
  - India: **1.1%**
  - Japan: **0.9%**
- Increases with age:
  - <65 years: **6.2%**
  - 75 years: **8.7%**

RISK OF PROGRESSION TO POAG

- About **1 in 10 (10%)** OHT patients develop glaucoma over 10 years.
- Majority **never develop glaucoma** in their lifetime.
- Based largely on OHTS (Ocular Hypertension Treatment Study) and EGPS.

PREDICTIVE FACTORS FOR CONVERSION (OHT → POAG)
(OHTS Multivariate Predictors — MOST IMPORTANT)

1. Intraocular Pressure (IOP)

- Strongest risk factor.
- Every **1 mmHg increase** increases risk by **~10%**.

2. Central Corneal Thickness (CCT)

- **POWERFUL predictor** of conversion.
- Thin CCT → underestimates true IOP + biomechanical susceptibility.

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- OHTS 5-year risk:
  - **CCT ≤ 555 µm → 3× higher risk**
  - **CCT > 588 µm → lowest risk**

3. Cup-to-Disc Ratio (C/D ratio)

- Larger C/D indicates structurally vulnerable disc or early undetected damage.
- Higher C/D → higher risk.

4. Pattern Standard Deviation (PSD)

- High PSD = higher risk (suggests early VF instability).

5. Age

- Older age strongly associated with progression.

6. Optic Disc Hemorrhage

- Increases glaucoma risk **2.6-fold**.

UNIVARIATE PREDICTORS (Less powerful)

- Race: Higher POAG risk in African descent.
- Males more likely to convert.
- Heart disease.
- Myopia (suspected, not OHTS significant).
- Family history (not significant in OHTS but supported by other studies).

INSIGNIFICANT FACTORS (OHTS)

- Diabetes
- Family history (unique OHTS result)
- Myopia (but clinically suspected to confer vulnerability)

OHTS KEY OUTCOMES

- **Early treatment lowers POAG risk by 50% at 10 years.**
- Absolute benefit greatest in **high-risk** individuals.
- Early treatment **not needed** in low-risk patients.
- Validated **risk calculators** available (OHTS + EGPS combined).

DIAGNOSIS OF OHT

A complete glaucoma evaluation is required:

1. Tonometry

- IOP >21 mmHg (repeated on different days, times).

2. Gonioscopy

- Must show **open angles** without secondary pathology.

3. Optic Disc Evaluation

- Normal RNFL and optic nerve appearance.
- No glaucomatous cupping.

4. Visual Fields (SAP)

- No glaucomatous defects.

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5. Pachymetry

- Measure CCT accurately (Orbscan®, Pachmate®).

6. OCT Imaging

- No RNFL thinning or macular GCIPL loss.
- Useful for detecting **pre-perimetric** glaucoma.

Other Useful Tests

- Corneal hysteresis
- Heidelberg Retina Tomography
- GDX scanning laser polarimetry

DIFFERENTIAL DIAGNOSIS

1. **Undiagnosed early POAG**
2. **Secondary causes of elevated IOP:**
    - Steroid-induced IOP rise
    - Angle recession
    - Angle-closure glaucoma (intermittent or latent)
    - Uveitic glaucoma

PRE-PERIMETRIC GLAUCOMA

- Structural glaucomatous optic disc or RNFL damage **without VF defects**.
- Detected on OCT, disc photos.
- Important because structural changes precede VF changes.

RISK ESTIMATION

Validated risk calculators use:

- Age
- CCT
- IOP
- PSD
- C/D ratio
- Disc hemorrhage
- Race

Goal: **5-year POAG risk (%)** estimation

- Treat if **annual risk ≥2%** (or **5-year risk ≥10%**).

MANAGEMENT

When to Treat OHT

Treat High-Risk Patients

Key indications:

- IOP **≥30 mmHg**
- Thin CCT (≤555 µm)
- Large C/D ratio

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- Disc hemorrhage
- High PSD
- Elderly patients with long life expectancy
- High calculated 5-year risk

Do NOT Treat Immediately

- Low-risk: young, thick CCT, borderline IOP.
- Low 5-year risk (<10%)
- May simply monitor.

TREATMENT OPTIONS

Same as POAG but less aggressive:

- Topical medications (PG analogues, beta-blockers, CAIs, etc.)
- SLT laser (alternative first-line in some patients)

OHTS showed:

- 20% IOP reduction target is effective
- Goal: IOP ≤24 mmHg
  (More aggressive targets in higher risk)

FOLLOW-UP & MONITORING

Low-risk OHT

- Yearly VF + OCT
- Disc photography

Moderate-risk

- Every 6–12 months

High-risk

- Every 3–6 months
- Consider treatment

COURSE & PROGNOSIS

- Progression rate: **1–2% per year** (average)
- Most patients remain stable long term.
- If glaucoma develops:
  - Structural changes often appear **before** VF changes.
- Treated high-risk individuals have excellent outcomes.

Exam Notes

- CYP1B1 → Congenital (primary congenital) glaucoma ("Child").
- MYOC (Myocilin) → Juvenile-onset open-angle glaucoma (JOAG) ("Young").
- OPTN (Optineurin) → Normal-tension glaucoma (NTG) ("Ocular Pressure Normal").
- LOXL1 → Pseudoexfoliation glaucoma ("LOX = exfoliation").
- WDR36 → Weakly associated with primary open-angle glaucoma (POAG).
- Normal episcleral venous pressure (EVP): 8–10 mmHg.

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NORMAL-TENSION GLAUCOMA (NTG)

Definition

Normal-Tension Glaucoma (NTG), also called low-tension or normal-pressure glaucoma, is a subtype within the spectrum of chronic open-angle glaucoma (COAG/POAG) characterized by:

- Progressive glaucomatous optic neuropathy
- Glaucomatous cupping with corresponding visual field defects
- **IOP consistently ≤21 mmHg on diurnal testing**
- **Open angles on gonioscopy**
- Absence of any secondary cause for optic nerve damage

NTG likely represents the low-IOP end of the POAG spectrum—IOP remains a pathogenic factor, but **non-IOP factors play a dominant role**.

Epidemiology

- Accounts for **16–50%** of POAG cases (population dependent)
- **More prevalent in Japanese/East Asian populations** (up to 75% of POAG cases)
- Typically presents in **patients >50–60 years**
- Females slightly more affected
- Left eye slightly more involved
- Genetic predisposition suggested

Pathogenesis

NTG is **multifactorial**, involving **IOP-dependent** and **IOP-independent** mechanisms.

1. IOP-Related Factors

- IOP contributes to damage **even in the high-normal range**
- Wide **diurnal IOP fluctuations**
- **Nocturnal IOP spikes** may be missed during clinic hours
- 30% reduction in IOP shown to slow progression (Low-Pressure Glaucoma Treatment Study)
- Some studies: worse field loss in eye with higher IOP
- Others: no relation (asymmetric IOP not predictive)

2. Vascular Factors

Evidence strongly implicates chronic vascular insufficiency:

- Optic nerve hypoperfusion
- **Disc hemorrhages** more common
- Increased **vascular resistance** in ophthalmic artery
- Reduced ocular blood flow and pulse amplitude
- Associated systemic vascular dysregulation:
  - Raynaud phenomenon
  - Migraines

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    - Nocturnal hypotension
    - Cardiovascular disease
    - Carotid occlusive disease
    - Cold-induced peripheral vasospasm
- Higher frequency of asymptomatic nocturnal myocardial ischemia

3. Systemic Blood Pressure Abnormalities

- Greater **nocturnal BP dips** (>20%)
- Episodes of hypotension or shock
- Over-treated systemic hypertension may worsen NTG

4. Hematologic Abnormalities

Reported associations:

- Increased blood/plasma viscosity
- Hypercoagulability (↑ platelet adhesiveness, prolonged euglobulin lysis time)
- Hypercholesterolemia
- Some studies show no significant coagulation differences

5. Autoimmune Mechanisms

- Higher prevalence of immune-related diseases
- Autoantibodies: anti-retinal proteins, anti-heat shock proteins
- Postmortem evidence of immunoglobulin deposition in retina

6. Structural/Lamina Cribrosa Factors

- Weak laminar connective tissue
- **Increased translaminar pressure gradients** due to:
    - Lower intracranial pressure (ICP)
    - Normal IOP → relatively higher translaminar difference
- Higher translaminar pressure correlates with field loss

7. Genetic Factors

- OPTN, OPA1, CDKN2B-AS polymorphisms
- Certain families show Mendelian patterns

8. Other Proposed Factors

- Mitochondrial dysfunction
- Obstructive sleep apnea
- Autonomic dysfunction
- Endothelial dysfunction (reduced flow-mediated vasodilation)

Risk Factors

- Age >50–60 years
- Female sex
- East Asian ethnicity
- Family history of glaucoma
- Thin CCT
- Vascular dysregulation (Raynaud, migraine)
- Nocturnal systemic hypotension

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- Myopia
- Previous AION or retinal vascular occlusion
- h/o migraine, peripheral vasospasm, chronic blood loss
- Thin body habitus, poor peripheral perfusion

Clinical Features

History

- Often asymptomatic
- Paracentral scotomas → early central visual involvement
- Systemic symptoms: migraines, cold extremities, hypotensive episodes
- Steroid history (risk of misclassified glaucoma)

Ocular Examination

Intraocular Pressure

- Usually high-normal (mid-to-high teens)
- Rarely low teens
- NTG patients may have **wider diurnal fluctuations**

Optic Nerve Head

- Glaucomatous cupping with:
  - **Thinner neural rim**, especially inferior/inferotemporal
  - More **localized RNFL defects**, closer to macula
  - **Disc hemorrhages more common**
  - Peripapillary atrophy common
  - Pallor **disproportionate to cupping** → consider non-glaucomatous causes
- Studies mixed on whether NTG discs differ morphologically from high-tension glaucoma

Retinal Nerve Fiber Layer

- More focal defects
- Closer to macula
- High-tension glaucoma → more diffuse defects

Visual Field Defects

- **Deeper, steeper, more localized** scotomas
- Frequently closer to fixation
- Superior hemifield commonly affected
- Some studies show faster progression
- Progression pattern:
  - NTG: area and depth increase proportionately
  - High-tension COAG: area increases first, depth later

Ocular Vascular Findings

- Focal arteriolar narrowing

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- Reduced perfusion pressure
- Abnormal color Doppler parameters
- Hypofluorescence on angiography

Investigations

- **Multiple IOP measurements**, diurnal curve
- Gonioscopy (open angles)
- Fundus exam & RNFL assessment
- Standard automated perimetry
- OCT RNFL / optic nerve head
- BP evaluation: 24-hr ambulatory monitoring
- Sleep studies if OSA suspected
- Neuroimaging:
    - Red flags: age <50, headaches, visual field respecting vertical midline, color vision loss, ONH pallor, neurologic signs

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Differential Diagnosis

1. Misclassified POAG / Secondary Glaucoma

- Undetected nocturnal or diurnal IOP spikes
- Previous high IOP now normalized
- Corticosteroid-induced glaucoma
- Pigmentary glaucoma improving with age
- Systemic medications lowering IOP (oral beta-blockers)

2. Non-Glaucomatous Optic Neuropathies

- Optic nerve anomalies: coloboma, pits, oblique insertion
- Kjer autosomal dominant optic atrophy
- AION (arteritic and non-arteritic)
- Optic neuritis
- Compressive optic neuropathies
- Methanol poisoning
- Traumatic optic neuropathy

3. Retinal Pathologies Mimicking Glaucoma

- BRAO
- Retinitis pigmentosa
- Chorioretinal lesions
- Cotton-wool spots

Management

1. Observation

- ~50% show minimal progression over 5–7 years
- Suitable for elderly or low-risk patients

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2. Lowering IOP (Mainstay)

Target: ≥30% reduction → mid-to-low teens or lower

Indicated if progression is documented.

Medical Therapy

- **Prostaglandin analogs**: first-line
- **Brimonidine**: possible neuroprotective effect
- **Beta-blockers**: avoid at bedtime (risk of nocturnal hypotension)
- **Carbonic anhydrase inhibitors (e.g., dorzolamide)**: may improve perfusion
- **Systemic calcium channel blockers**: improve vasospasm & optic nerve perfusion in selected patients

Laser

- **Selective Laser Trabeculoplasty (SLT)** — modest IOP reduction

Surgery

- **Trabeculectomy with MMC**
- Needed when progression continues despite low pressures
- Goal: Single-digit IOPs
- Caution: postoperative hypotony risk higher in NTG

3. Address Vascular Factors

- Treat systemic hypertension / hypotension appropriately
- Adjust nighttime antihypertensive dosing
- Head-up sleeping position
- Manage migraine or Raynaud's disease
- Evaluate for OSA

---

4. Lifestyle & Adjunctive Measures

- Avoid prolonged head-down positions
- Good hydration
- Consider supplements (limited evidence): Ginkgo biloba, resveratrol
- Avoid excessive nighttime BP drops
- Avoid smoking

Prognosis

- NTG progression often slow but persistent
- Poor prognostic indicators:
  - Female sex
  - Migraine
  - Optic disc hemorrhage
- Long-term outcomes:
  - Unilateral blindness: ~10% over 20 years
  - Bilateral blindness: ~1–2% with treatment
- Properly lowered IOP significantly slows progression but does not eliminate risk

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ANTIGLAUCOMA MEDICATIONS

INTRODUCTION

Intraocular pressure (IOP) depends on:

1. Rate of aqueous humor production
2. Trabecular (conventional) outflow
3. Uveoscleral (unconventional) outflow
4. Episcleral venous pressure

Lowering IOP is the only proven method to prevent glaucoma progression.

MECHANISM-BASED CLASSIFICATION

1. Drugs Increasing Aqueous Outflow

- **Uveoscleral:** Prostaglandin analogues, EP2 agonists --- 25-35%
- **Trabecular:** Cholinergics, ROCK inhibitors, Nitric oxide donors

2. Drugs Decreasing Aqueous Production

- β-blockers --- 20-30%
- Carbonic anhydrase inhibitors --- 15-20%
- α_2-adrenergic agonists --- 15-20%

3. Drugs Reducing Vitreous Volume

- Hyperosmotic agents

Q.Disadvantages of Using Two Separate Drugs

- Poor compliance
- Difficult dosing schedule
- Washout effect
- Increased preservative toxicity
- Higher cost

Q.Advantages of Fixed Combinations

- Simple dosing
- Better compliance
- Avoids washout effect
- Greater IOP reduction
- Less preservative exposure
- Reduced systemic toxicity
- More economical

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I. PROSTAGLANDIN ANALOGUES (PGAs)

Class

- PGF2α derivatives / Prostamides / Eicosanoids

Drugs

- **Latanoprost** 0.005% – once daily (qHS)
- **Travoprost** 0.004% – qHS – *good effect over diurnal variation & BAK free formulation*
- **Bimatoprost** 0.01% / 0.03% – qHS
- **Tafluprost** 0.0015% – qHS
- **Unoprostone** 0.15% – BID
- **Latanoprostene bunod** 0.024% – qHS

Mechanism of Action (PG Depth)

- FP receptor agonists in: Ciliary muscle & Sclera
- ↑ Matrix metalloproteinases (MMP-1, MMP-9)
- ↓ Collagen types I & III
- **Extracellular matrix remodeling**
- ↑ Intermuscular spaces → ↑ **uveoscleral outflow**

Latanoprostene bunod 0.024%

- Releases nitric oxide (NO)
- NO → guanylate cyclase → ↑ cGMP
- Relaxes trabecular meshwork + Schlemm canal
- **Dual outflow enhancement**

IOP Reduction

- **25–35%** (maximum among topical drugs)
- Bed Time – *max efficacy & decreased ADR d/t vasodilation*
- Flat diurnal curve
- Peak: 10–14 hours
- Full effect: 4–6 weeks

Indications

- Primary open-angle glaucoma (POAG)
- Ocular hypertension
- Normal-tension glaucoma (NTG)
- Secondary open-angle glaucoma
- Selected chronic angle-closure cases

Contraindications (Relative)

- Active uveitis (↑ inflammation)
- Hypersensitivity
- Herpetic keratitis (reactivation)
- Aphakia / posterior capsular rent → CME

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- Recent intraocular surgery
- Pregnancy (relative)—*1st T: abortion & 3rd T: Induction of labor*
- Contact Lens wear

Adverse Effects

Ocular

- Conjunctival hyperemia
- Iris pigmentation
- Eyelash hypertrichosis
- Fornix shortening
- Periorbital fat atrophy (PAP)
- Rare CME, anterior uveitis (Iritis)
- Reactivation of viral keratitis (HSV)

Systemic

- No systemic ADR
- Headache & myalgia (rare)

PG Exam Pearls

- **First-line drug for POAG**
- Best nocturnal IOP control
- Once-daily bedtime dosing
- Switch within PG class if non-responder
- Remove contact lenses before instillation
- **DOC in uveitic glaucoma**: Timolol, Brimonidine, Dorzolamide
- **Benefits of PG**: Single dosing, 30- 35% IOP reduction, Flat IOP curve, Nil systemic ADR
- **Ways to decrease systemic absorption:** Punctal Occlusion, Closing eyes for 3 minutes
- **Advantages of Dorzolamide–Timolol (DT)**
    - Greater IOP reduction than either drug alone
    - Additive/synergistic effect
    - Better efficacy
    - No electrolyte imbalance (unlike systemic CAIs)

Indications for Surgery in Glaucoma

- Failure to achieve target IOP.
- Progressive glaucomatous damage despite maximal medical therapy (3 drugs for ≥3 months).
- Drug intolerance.
- Poor compliance with medical therapy.

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II. BETA-ADRENERGIC BLOCKERS

Class

- Non-selective and β_1-selective blockers

Drugs

Cardioselective β_1-Blocker

- Betaxolol 0.25–0.5% – BD *(β_1-selective)*

Non-Selective β-Blockers

- Timolol 0.25–0.5% – BD
- Levobunolol 0.25–0.5% – OD/BD
- Carteolol 1% – BD

Ideal Time: Morning – *blunt early morning increased IOP d/t circidian rhythm*

Mechanism of Action

- Block β-receptors in non-pigmented ciliary epithelium
- ↓ Adenyl cyclase → ↓ cAMP
- ↓ Na^+/K^+ ATPase activity
- **↓ aqueous humor production**

IOP Reduction: 20–30%, Reduced nocturnal efficacy

Indications

- POAG, Secondary glaucomas, Add-on to PGAs

Contraindications

- Bronchial asthma, COPD
- Sinus bradycardia
- Heart block
- Congestive heart failure
- Diabetes mellitus
- Peripheral vascular disease
- Myasthenia gravis

Adverse Effects

Ocular

- Dry eye, Superficial punctate keratitis, Corneal anesthesia

Systemic

- Bradycardia
- Hypotension
- Bronchospasm
- Depression, fatigue
- Masked hypoglycemia

PG Exam Pearls

- Avoid bedtime dosing (systemic hypotension)
- Betaxolol safer in pulmonary disease (less potent)
- Long-term drift & short-term escape phenomenon

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III. ALPHA-2 ADRENERGIC AGONISTS

Drugs

- **Brimonidine** 0.1–0.2% – TDS— ***Alphagan-Z (0.1%),Alphagan-P (0.15%),Alphagan (0.2%)***
- **Apraclonidine** 0.5–1% – TDS (short-term) – *decrease aqueous production*

Mechanism of Action

- Presynaptic α_2 stimulation:
  - ↓ norepinephrine release
  - ↓ aqueous production
- Postsynaptic α_2:
  - ↑ uveoscleral outflow
- Possible **neuroprotective effect**

IOP Reduction: 20–25%

Indications

- POAG (adjunct)
- Post-laser IOP spike prevention (apraclonidine)

Contraindications

- Infants & children <2 years--- *crosses BBB—Somnolence, Bradycardia, Hypotension, Apnea, CNS depression*
- MAO inhibitors
- Tricyclic antidepressants
- Severe cardiovascular disease

Adverse Effects

- Allergic follicular conjunctivitis
- PEEs, decreased Tear production, decreased corneal sensation
- Dry mouth
- Fatigue
- Hypotension
- Apnea & CNS depression in infants
- Somnolence

PG Exam Pearls

- Apraclonidine → tachyphylaxis
- Brimonidine-Purite better tolerated
- Preferred short-term adjunct

IV. CARBONIC ANHYDRASE INHIBITORS (CAIs)

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A. TOPICAL CAIs

- **Dorzolamide** 2% – TDS
- **Brinzolamide** 1% – TDS

Mechanism

- Inhibit CA-II in ciliary processes
- ↓ bicarbonate → ↓ Na^+ transport
- ↓ aqueous production

IOP Reduction

- **15–20%**

B. SYSTEMIC CAIs

- **Acetazolamide** 250 mg QID / 500 mg SR BD --- *avoided in Sickle cell—low pH causes more sickling*
- **Methazolamide** 50 mg BD–TDS

IOP Reduction

- **30–40%**

Indications

- Acute angle-closure glaucoma
- Pre-operative IOP control
- Refractory glaucoma

Contraindications

- Sulfonamide allergy
- Renal failure
- Chronic Liver Disease
- Renal Transplant
- Addison's disease
- Pregnancy

Adverse Effects

- **Ocular:** Corneal edema, transient myopia, burning/stinging.
- **Metabolic: Hyperchloremic metabolic acidosis**, hypokalemia, paresthesia.
- **GU: Renal calculi**, nocturia, impotence.
- **GI:** Nausea, vomiting, abdominal pain, GI upset.
- **Hemato(rare): Aplastic anemia**, agranulocytosis, neutropenia, thrombocytopenia.
- **CNS:** Fatigue, Tingling sensation, drowsiness, headache, paresthesia.
- **Derma (sulfonamide reactions):** Rash, pruritus, Stevens–Johnson syndrome (SJS), toxic epidermal necrolysis (TEN), exfoliative dermatitis.

PG Exam Pearls

- Most potent aqueous suppressors
- Topical CAIs not additive to systemic CAIs
- Avoid long-term systemic use

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V. CHOLINERGIC AGONISTS (MIOTICS)

Drugs

- **Pilocarpine** 0.5–4% – QID (gel qHS)
- **Carbachol** 1.5% – TDS
- **Echothiophate iodide** – BD

Mechanism of Action

- **POAG:** M_3 receptor stimulation → Ciliary muscle contraction (longitudinal fibers) → Scleral spur traction → Trabecular meshwork opens (↑ porosity) → ↑ Aqueous outflow via Schlemm's canal → ↓ IOP.
- **PACG:** M_3 receptor stimulation → Miosis → Peripheral iris pulled away from trabecular meshwork → Relieves pupillary block → Opens anterior chamber angle → ↑ Aqueous outflow → ↓ IOP.

IOP Reduction: 20–25%

Indications

- Acute angle-closure glaucoma
- Plateau iris syndrome
- Post-laser prophylaxis

Contraindications

- Uveitis
- High myopia
- Phacolytic glaucoma
- Cataract
- Neovascular glaucoma
- Retinal detachment risk

Adverse Effects

Ocular

- Miosis → poor night vision
- Brow ache
- Induced myopia – *d/t CB contraction*
- Lacrimation—*d/t punctal stenosis*
- Retinal detachment (rare)

Systemic

- Bradycardia
- Bronchospasm
- GI cramps

PG Exam Pearls

- Oldest antiglaucoma drug
- Mainly for **acute angle closure**
- Pilocarpine 0.1% diagnostic for Adie pupil

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VI. RHO-KINASE (ROCK) INHIBITORS

Drugs – Dose

- **Netarsudil** 0.02% – OD
- **Ripasudil** 0.4% – BD
- **Netarsudil + Latanoprost** – OD

Mechanism of Action

- Inhibit ROCK-1 & ROCK-2
- ↓ Actin stress fibers
- Relax trabecular meshwork
- ↑ Schlemm canal permeability
- ↓ Episcleral venous pressure

IOP Reduction

- **20–30%**

Adverse Effects

- Conjunctival hyperemia (most common)
- Subconjunctival hemorrhage
- Corneal verticillate

(*Drug causing Verticillate: Amiodarone, HCQ, Chloroquine, Tamoxifen, Indomethacin, RhoKinase Inhibitor*)

PG Exam Pearls

- Only drugs acting directly on **trabecular cytoskeleton**
- Useful in resistant glaucoma

Newer Ocular Hypotensive Agents

- Natural cannabinoids.
- ECM hydrolysis activators (MMPs – Matrix Metalloproteinases) → ↑ Trabecular outflow.
- Cytoskeleton modulators → Relax trabecular meshwork.
- Ethacrynic acid → Increases trabecular outflow.
- Protein kinase inhibitors → Increase aqueous outflow.
- cGMP-enhancing compounds → Improve aqueous outflow.

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VII. HYPEROSMOTIC AGENTS

Drugs – Dose

- **IV Mannitol 20% IV** – 1–2 g/kg over 30–60 min --- *less irritating for blood vessel & can be used in DM & CRFpatients.*
  (Instruction: no to get up immediately after injection—causes hypotension)
- **Oral Isosorbide 45%**
- **Oral Glycerol 50%**– 1–1.5 g/kg

Mechanism

- ↑ Plasma osmolarity
- Fluid shift from vitreous → plasma
- ↓ Vitreous volume
- Rapid IOP reduction

ADR

- **Ocular**: Rebound rise in IOP, intraocular hemorrhage.
- **CNS**: Hyperosmolarity causing confusion, disorientation, thirst, chills, and fever.
- **GI**: Nausea, vomiting, diarrhea, and abdominal cramps.
- **Renal/Fluid & Electrolytes**: Diuresis leading to dehydration, hypovolemia, and electrolyte imbalance.
- **CVS**: Angina, pulmonary edema, and congestive heart failure (CHF/CCF).
- **Others**: Hyperglycemia and hypersensitivity reactions.

Indications

- Acute angle-closure glaucoma
- Pre-operative IOP lowering
- Malignant Glaucoma

Contraindications

- Cardiac failure
- Renal failure
- Anuria
- Pulmonary edema
- Dehydration
- Diabetes (glycerol)

PG Exam Pearls

- Emergency drugs
- Short-acting
- Risk of rebound IOP rise

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Neuroprotective Drugs in Glaucoma

Prevents retinal ganglion cell (RGC) death independent of IOP reduction.

- ❖ **NMDA Receptor Antagonist**
  - **Memantine:** Blocks glutamate-mediated excitotoxicity → ↓ Ca^{2+} influx → Prevents RGC apoptosis.
- ❖ **Nitric Oxide (NO) Synthase Inhibitor**
  - **Aminoguanidine:** Inhibits NO-mediated neurotoxicity.
- ❖ **Calcium Channel Blockers**
  - **Nimodipine**, Verapamil: ↑ Optic nerve blood flow; useful in normal-tension glaucoma (NTG); enhance RGC survival.
- ❖ **Other Neuroprotective Agents**
  - **Brimonidine (α_2-agonist):** Neuroprotective effect beyond IOP lowering.
  - **Neurotrophic factors: BDNF, CNTF, NGF** → Promote RGC survival.
  - **Antioxidants: Resveratrol, α-lipoic acid** → Reduce oxidative stress.
  - **Apoptosis inhibitors:** Prevent programmed RGC death.
  - **Caspase inhibitors:** Block apoptosis pathway.
  - **Coenzyme Q10:** Mitochondrial neuroprotection.

Newer Drug Delivery Systems in Glaucoma

- **Durysta® (Bimatoprost intracameral implant):** Biodegradable implant; sustained release **3–4 months**; **FDA-approved (2020)** for **POAG/OHT**.
- **iDose® Travoprost:** Intracameral implant with **>6 months** sustained release; **~32–33% IOP reduction**.
- **Bimatoprost ring: Preservative-free forniceal ring** containing **13 mg bimatoprost**.
- **Travoprost punctal plug:** Inserted into the **inferior canaliculus** for sustained drug delivery.
- **Drug-eluting contact lens: Silicone hydrogel lens** loaded with nanoparticles for continuous ocular drug release.

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NEOVASCULAR GLAUCOMA (NVG)

Neovascular glaucoma (NVG) *aka 100 days Glaucoma/ Rubeotic Glaucoma* is a **secondary glaucoma caused by aggressive neovascularization of the iris (rubeosis iridis) and anterior chamber angle**, leading to:

- Formation of a **fibrovascular membrane**
- Progressive **synechial angle closure**
- **Marked elevation of IOP**
- Rapid progression to **optic nerve damage** and **painful blind eye** if untreated.

Epidemiology

- Accounts for **~3–4% of severe glaucoma cases**.
- Most common causes: **Ischemic CRVO**, **proliferative diabetic retinopathy (PDR)**, **ocular ischemic syndrome**.
- Bilateral disease is rare but **risk to fellow eye is high** because underlying systemic disease usually affects both eyes.

Theories of Neovascularization

1. **Retinal Hypoxia Theory** (Most Accepted)
   Retinal ischemia → ↑ VEGF (± bFGF, TGF-α, TNF-α) → NVI & NVA → NVG.
2. **Angiogenic Factor Theory**
   Tumor/ischemic retina → Release of angiogenic factors → Neovascularization.
3. **Chronic Vasodilatation Theory**
   Chronic iris hypoxia → Persistent iris vasodilatation → Neovascularization.
4. **Loss of Inhibitory Factors Theory**
   Loss of anti-angiogenic factors after PPV/cataract surgery → Unopposed angiogenesis → Neovascularization

Pathogenesis

Underlying mechanism

- **Retinal ischemia → ↑ VEGF & angiogenic factors → neovascularization of iris and angle.**
- New vessels form a **fibrovascular membrane** that:
  - Covers trabecular meshwork
  - Contracts → **peripheral anterior synechiae (PAS)**
  - Causes **irreversible angle closure** & very high IOP

Key drivers

- **VEGF (main mediator)**
- Inflammatory cytokines (IL-6, IL-8)
- Hypoxia-related factors (HIF-1α)

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Stages (Kahook)

1. **Pre-rubeosis Stage:** NVD, NVE
2. **Pre-glaucoma stage (rubeosis iridis)**
   - NVI at pupillary margin; IOP normal.
3. **Open-angle NVG**
   - NVA present; fibrovascular membrane impairs trabecular outflow; IOP ↑.
4. **Closed-angle NVG**
   - Membrane contraction → 360° PAS → very high IOP & pain.
5. **Burnt out Stage**

Wand's Classification

- **Stage I:** New vessels at the pupillary margin**.**
- **Stage II:** New vessels extend to the iris collarette.
- **Stage III:** New vessels involve the anterior chamber angle (NVA).
- **Stage IV:** New vessels cross the scleral spur (SS) → Advanced angle involvement.

Etiology / Causes

1. Vascular Causes (Most Common)
- Diabetic retinopathy (MC cause)
- CRVO (Most common retinal vascular cause)
- CRAO
- BRVO
- BRAO

2. Inflammatory Causes
- Chronic uveitis
- Chronic retinal detachment
- Sympathetic ophthalmia (SO)
- VKH
- Endophthalmitis

3. Neoplastic Causes
- Malignant melanoma
- Retinoblastoma
- Ocular metastasis

4. Systemic Diseases
- Diabetes mellitus
- Sickle cell disease
- SLE

5. Vascular Disorders
- Carotid artery obstruction (Ocular ischemic syndrome)
- Carotid-cavernous fistula (CCF)
- Giant cell arteritis (GCA)
- AV malformation (AVM)

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6. Miscellaneous

- ROP
- PHPV/PFV
- FEVR
- Coats disease
- Retinoschisis

7. Surgical / Iatrogenic

- PPV / Vitrectomy
- Retinal detachment surgery
- Cataract extraction
- Radiation
- Nd:YAG capsulotomy

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5. Clinical Features

Symptoms

- Range from asymptomatic → **severe pain**, redness
- Reduced vision, photophobia

Signs

Cornea

- Corneal edema when IOP markedly elevated

Anterior Chamber

- Cells, flare, posterior synechiae
- Sometimes hyphema ("blood in AC")

Iris

- **Earliest hallmark: fine new vessels at pupillary margin (rubeosis).—***1st sign (Inv: increased blood vessel permeability at pupillary margin)*
- Vessels grow radially toward the angle.
- May join dilated collarette vessels.
- Ectropion Uvea: d/t radial traction (*contraction of fibrovascular membrane in angle & iris)*

Angle (Gonioscopy)

- Very important diagnostic step
- Early **NVA** often subtle
- Proliferation of **fibrovascular membrane** → PAS → 360° angle closure
- Late stage: extremely high IOP, ciliary body shutdown → hypotony (rare)

Lens

- Cataract is common with chronic ischemia

Posterior Segment

- Depends on cause: PDR, CRVO, OIS, chronic RD, tumors --- *NVD, NVE*
- Optic nerve cupping in advanced disease

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6. Investigations

- **Fluorescein angiography (FA):** confirms retinal ischemia, non-perfusion
- **B-scan US:** when media opaque; rule out RD or tumor
- **Anterior segment OCT:** angle assessment
- **Ocular Doppler:** if OIS suspected
- **Systemic investigations:** diabetes, carotid disease, hypertension

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7. Treatment

Goals

1. Treat underlying ischemia
2. Control neovascularization
3. Lower IOP
4. Preserve comfort and vision when possible

A. Control of Neovascularization

1. Panretinal Photocoagulation (PRP) – Mainstay

(Laser T/t in NVG: Ant Segment: Goniophotocoagulation, Post Segment: PRP, TSCP)

- Most effective to induce **regression of NVI/NVA**
- Prevents progression to NVG when applied early
- Will **NOT** reverse established PAS
- If view is hazy:
  - Indirect laser under anesthesia (LIO)
  - Use of iris hooks
  - Trans-scleral cryotherapy (opaque media)

2. Anti-VEGF Therapy (Bevacizumab / Ranibizumab / Aflibercept)

- Dose: **Bevacizumab 1.25 mg/0.05 ml**
- Routes: intravitreal or intracameral
- Rapid regression of vessels (within days)
- Good for:
  - Pain relief
  - Pre-PRP stabilization
- Effects temporary → repeated injections often required
- In ischemic CRVO: can **delay NVG onset up to 18 months**

B. IOP-Lowering Therapy

Medical *(IOP <40)*

- Treat as POAG **but avoid miotics** (worsen inflammation & PAS)
- **Useful agents:**
  - Beta blockers
  - Alpha-agonists (apraclonidine)

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- Carbonic anhydrase inhibitors (topical; oral acetazolamide cautiously in diabetics)

- **Topical steroids** for inflammation
- **Atropine 1% BID** to reduce pain & prevent synechiae, increase uveoscleral outflow

Drugs C/I in NVG: *Miotics, Epinephrine, PG*

C. Surgical Management

1. Glaucoma Drainage Devices (GDDs)

- Preferred when vision ≥ hand movements
- Tubes work better in inflamed eyes
- Risks: hypotony, tube blockage, hyphema (common)

2. Trabeculectomy

- Only if inflammation controlled & anti-VEGF given pre-op
- Usually with MMC & postoperative 5-FU
- Higher failure rate than GDDs

3. Cyclodestructive Procedures

- **Cyclodiode laser** most common
- Traditionally used in eyes with poor visual potential
- Now used earlier to:
  - Reduce IOP
  - Clear corneal edema to allow PRP
- Avoid overtreatment (risk of hypotony)

4. Pars Plana Vitrectomy with Endolaser

- In diabetic tractional RD or non-clearing vitreous hemorrhage
- Helps deliver PRP and reduce ischemic drive

5. Pain-Relief Procedures (End-Stage Eye)

- Retrobulbar alcohol (risk: ptosis)
- **Enucleation or evisceration** if blind painful eye persists

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8. Follow-Up

Critical during high-risk periods:

- First **2–3 months after ischemic CRVO**
- First **few weeks after diabetic vitrectomy**

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9. Prognosis

- Visual prognosis often poor once PAS-induced closure occurs.
- **25–50% become blind** despite treatment.
- **Life expectancy reduced**—patients live ~50% of expected lifespan due to associated systemic vascular disease.
- Good presenting VA predicts better survival and visual outcomes.

Goal in advanced NVG is often a "blind but comfortable eye."

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Differential Diagnosis of NVG

1. Acute congestive stage of Primary Angle-Closure Glaucoma (PACG)
2. Uveitic glaucoma
3. Phacolytic glaucoma
4. Iridocorneal Endothelial (ICE) syndrome
5. Old ocular trauma
6. Lens-induced glaucoma
7. Fuchs' heterochromic iridocyclitis (Fuchs uveitis syndrome)

Complications of NVG

- **Painful bullous keratopathy** → Due to chronic uncontrolled elevated IOP.
- **Complete synechial angle closure** → Permanent closure by fibrovascular peripheral anterior synechiae (PAS).
- **Intractable glaucoma** → Refractory high IOP despite maximal medical and surgical treatment.

Feature	**NVI / NVA**	**Normal Iris Vessels**
Location	At pupillary margin and angle	Normal iris vasculature
Length	Short vessels	Longer vessels
Pattern	Irregular	Regular
Caliber	Thin	Normal caliber
Course	Tortuous	Radial
Branching	Arborizing	Non-arborizing
Fenestration	Fenestrated	Non-fenestrated
Scleral spur (SS)	Crosses scleral spur	Does not cross scleral spur
FFA	Leaks fluorescein	No leakage
Histology	Single layer of endothelial tube (no mature vessel wall)	All three vessel wall layers present
Clinical significance	Poor prognosis; indicates active neovascularization	Intact, normal iris vasculature

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Newer Treatment of Neovascular Glaucoma (NVG)

- **Anti-VEGF therapy**: Produces rapid regression of iris (NVI) and angle (NVA) neovascularization.
- **α-Interferon therapy**: Anti-angiogenic effect.
- **Troxerutin**: anti-angiogenic and antioxidant properties.
- **Gene therapy targeting the PEDF gene**

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STERIOD-INDUCED GLAUCOMA

INTRODUCTION

Steroid-induced glaucoma (SIG) is a **secondary open-angle glaucoma** caused by topical, periocular, intraocular, systemic, or endogenous corticosteroids (e.g., Cushing's syndrome).

About **one-third of the general population** shows some IOP elevation ("steroid responders").

The rise in intraocular pressure (IOP) is due to **increased resistance to aqueous outflow** at the **trabecular meshwork (TM)**.

Typical onset:

- **Topical steroids:** 2–4 weeks
- **Intravitreal triamcinolone:** lasts 2–4 months
- **Dexamethasone implant (Ozurdex®):** up to 6 months

SIG clinically mimics **primary open-angle glaucoma (POAG)** with an open angle and gradual asymptomatic IOP elevation.

Acute presentations are rare but may occur after intensive systemic steroid therapy.

ETIOPATHOGENESIS

Steroids cause structural, functional, and biochemical changes in the trabecular meshwork, leading to reduced aqueous drainage.

1. Genetic Basis

Steroid responsiveness appears genetically determined.

Population response after 6 weeks of steroid therapy:

- **High responders (5%)**: IOP ↑ >15 mmHg *(ie IOP >30)*
- **Moderate responders (35%)**: IOP ↑ 6–15 mmHg *(ie IOP 22 to 30)*
- **Non-responders (60%)**: IOP ↑ <6 mmHg *(ie IOP: WNL)*

Prevalence in POAG patients / relatives:

- POAG patients: 90% respond
- POAG siblings: 70%
- POAG offspring: 70%

Responder = higher lifetime risk of developing POAG.

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2. Pathophysiological Mechanisms

A. Glycosaminoglycan (GAG) Theory

- Steroids inhibit lysosomal hydrolases.
- GAGs cannot depolymerize → become hydrated → **trabecular space narrows** → outflow resistance ↑.

B. Endothelial Cell / Phagocytosis Theory

- TM endothelial cells normally phagocytose debris.
- Steroids suppress phagocytosis → debris accumulates → blockage of TM → IOP elevation.

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C. Prostaglandin Theory

- Steroids inhibit prostaglandin E & F synthesis.
- Prostaglandins normally ↑ uveoscleral outflow → inhibition causes IOP rise.

D. Trabecular Meshwork (TM) Cellular Effects

- Activation and enlargement of TM cells
- Increased TM cell & nuclear size
- Cytoskeletal rigidity
- Reduced gap junction function

E. Extracellular Matrix Changes

- Increased ECM deposition
- ↑ Fibronectin, collagen, elastin
- Thickened TM beams
- Altered GAG expression → increased outflow resistance

F. Gene Expression

- Upregulation of Myocilin & Optineurin genes
- ↑ MYOC gene (TIGR protein) in TM — same gene implicated in POAG
- ↓ MMPs → impaired ECM turnover

RISK FACTORS

Patients at highest risk include:

- Primary open-angle glaucoma / ocular hypertension
- Family history of glaucoma
- High myopia
- Diabetes mellitus
- Connective tissue diseases (e.g., rheumatoid arthritis)
- Traumatic angle recession
- Young age (children are very susceptible)
- Adrenal hyperplasia / Cushing's syndrome
- Long duration or high potency steroid use

CLINICAL FEATURES

Typical Presentation (Chronic SIG)

Resembles POAG:

- Asymptomatic in early stages
- Elevated IOP
- White, quiet eyes
- Open angle on gonioscopy
- Glaucomatous optic nerve cupping
- Visual field defects after long-standing elevation

Acute Presentation

Rare but possible after intensive steroid exposure:

- Sudden marked IOP rise

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- Corneal edema
- Pain, headache, blurred vision

POTENCY OF VARIOUS STEROIDS

(General principles)

- Higher anti-inflammatory potency → higher IOP-elevating risk

Examples:

- **High potency:** Dexamethasone, prednisolone acetate 1%
- **Moderate:** Rimexolone, fluorometholone
- **Low-risk soft steroids:** Loteprednol etabonate

MANAGEMENT

1. Prevention

- Use steroids **judiciously** and at **lowest effective dose**.
- Prefer **low-IOP-risk steroids** when possible (loteprednol, fluorometholone).
- Regular IOP monitoring for patients on any steroid >2 weeks.

---

2. Discontinue / Reduce Steroid Use

First-line treatment.

- IOP often normalizes within **10 days–4 weeks**.
- Faster in acute form (days), slower in chronic form.

3. Substitute With a Safer Steroid

Examples:

- Loteprednol etabonate
- Fluorometholone 0.1%
- Rimexolone 1%

---

4. Medical Therapy for IOP Control

Same as POAG except **avoid miotics**.

Effective drugs:

- Beta-blockers (timolol)
- Carbonic anhydrase inhibitors
- Alpha-agonists
- Prostaglandin analogs (variable efficacy depending on TM status)

Timolol 0.5% is very effective during steroid washout.

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5. Persistent IOP Elevation

Occurs in ~3% of patients even after stopping steroids.

More common in those with:

- Strong family history of glaucoma
- Long duration of steroid therapy

Management:

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- Long-term medical therapy
- Filtration surgery (trabeculectomy)
- Glaucoma drainage implant
- Removal of steroid depot:
    - Excision of subtenon depot
    - Removal of intravitreal implant
    - Pars plana vitrectomy for retained intraocular steroid material

---

6. Emerging / Investigational Treatments

- **Anecortave acetate:** non-glucocorticoid cortisone derivative under study; may reduce steroid-induced ocular hypertension.

KEY CLINICAL POINT

Any patient on topical steroids with elevated IOP should have the steroid stopped immediately if possible.
A rapid fall in IOP confirms the diagnosis of a “steroid responder.”

SUMMARY

Steroid-induced glaucoma is a preventable and treatable condition.

- Onset typically 2–4 weeks after steroid initiation.
- Caused by steroid-induced TM dysfunction, ECM accumulation, and genetic susceptibility.
- Risk highest in POAG patients, family history, children, and high myopes.
- Discontinuing steroids is the most effective treatment.
- Persistent cases may require medical or surgical intervention.

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PRIMARY ANGLE-CLOSURE GLAUCOMA

Definition:

- Occlusion of the trabecular meshwork by the peripheral iris (iridotrabecular contact), obstructing aqueous outflow.
- Types:
  - Primary: Occurs in anatomically predisposed eyes.
  - Secondary: Caused by other ocular or systemic factors.

Etiopathogenesis

Predisposing Factors

Anatomical: "SHM-SPA"

- **S**hort axial length
- **H**yperopia
- **M**icrocornea
- **S**hallow AC
- **P**lateau iris
- **A**nteriorly placed lens / Microspherophakia

Physiological: "MDNP"

- **M**id-dilated pupil
- **D**im illumination
- **N**ear work
- **P**rone position

Demographic: "AFFR-T"

- **A**ge ↑ *(Increased Age: Increase Lens Thickness, Anterior position of lens, miotic pupil)*
- **F**emale
- **F**amily history
- **R**ace (Asian/Indian/Inuit; Africans also at increased risk)
- **T**ype A personality

High-risk biometric cut-offs:

- Axial length < 22 mm
- AC depth < 2.2 mm
- Lens thickness > 4 mm
- Lens thickness / axial length ratio > 20%

B. Mechanisms of IOP Elevation

- Most cases involve multiple mechanisms

Pupillary Block (~70%)

- Mid-dilation → **relative block** → posterior pressure → **iris bombe** → angle closure

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- **Triggers:**
  - Dark environment, stress
  - Prone position, Valsalva
  - Mydriatics: **phenylephrine, tropicamide, atropine**
  - **Pilocarpine** may worsen early attacks (lens moves forward)

Plateau Iris

- Anatomically anterior position of CB
- **Normal central AC depth**, but **peripheral crowding**
- Acute attacks can occur **even with patent PI**
- **UBM** for diagnosis – *double hump sign*
- **Treatment:** Miotics + **Laser Iridoplasty**
- **Pseudoplateau iris:** iris/ciliary body cysts → treat with **YAG/needle cyst puncture** ± iridoplasty

Lens-Induced

- **Thick / intumescent / anteriorly positioned lens** → reduces angle width
- **Lens extraction** may be curative in lens-dominant PACG

Gonioscopy

Gold standard for:

- Confirming angle closure
- Distinguishing appositional vs. synechial closure (**Indentation**)

Supporting Methods:

- **Shaffer grading** (angle width)

Grade	Angle	Structures Seen	Notes
4	35–45°	Ciliary body (SL+TM+SS+CB)	Widest; myopia, pseudophakia
3	25–35°	Scleral spur (SL+TM+SS)	Open angle
2	~20°	Trabeculum (SL+TM)	Narrow angle
1	~10°	Schwalbe line (SL)	Very narrow
Slit	–	None visible	Angle present but unclear
0	0°	None	Closed angle

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- **Van Herick** (screening)

Grade	PAC / CT	Description	Comment
4	≥1	PAC ≥ CT	Wide open
3	¼–½	PAC ¼–½ CT	Incapable of closure
2	¼	PAC ≈ ¼ CT	Should undergo gonioscopy
1	<¼	PAC < ¼ CT	Dangerously narrow; gonioscopy needed

- **Spaeth system** (detailed configuration)
  Superior quadrant is typically **narrowest** → must inspect carefully

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Key Investigations

- Gonioscopy: diagnosis & prognosis
- UBM / AS-OCT: identify mechanism
- Optic nerve & visual field: PACG staging

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CLASSIFICATION OF PRIMARY ANGLE-CLOSURE DISEASE

A) Based on Clinical Presentation

1. **Latent** PACG → corresponds to **Primary Angle-Closure Suspect (PACS)**
2. **Subacute** (Intermittent) PACG → corresponds to **Subacute PAC**
3. **Acute** PACG → corresponds to **Acute PAC**
4. **Chronic** PACG → corresponds to **Primary Angle-Closure Glaucoma (PACG)**

B) Based on Pathophysiology

Due to apposition of iris to TM at different levels:

1. **Pupillary block** ACG (most common)
2. **Plateau iris** ACG
3. **Phacomorphic** ACG
   - Lens-related / Lens block

C) ISGEO Classification (2006) Based on Natural History

Stage	Gonioscopy	Optic Disc	IOP/PAS	Symptoms
PACS	ITC > 180°; no PAS	Normal	IOP normal; PAS absent	**No symptoms**
PAC	ITC > 180°	Normal	IOP ↑ and/or PAS present	± Symptoms
PACG	ITC > 180°	**Glaucomatous ONH & VF damage**	IOP ↑ ± PAS	Variable

→ **Term PACD** includes all 3 stages.

CLINICAL PRESENTATIONS

Primary Angle-Closure Suspect (PACS)

- **Symptoms:** None
- **Typical detection:** routine exam, fellow eye of acute attack, screening
- **Suspicious signs:**
  - Shallow AC, convex iris-lens diaphragm
  - **Van Herick grade ≤ 2**
  - **Eclipse sign** +
- **IOP & Optic nerve:** Normal
- **Risk:** may progress to PAC or PACG

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Management

- **Prophylactic Laser PI** if >270° ITC or fellow eye of APAC
- Patient education + periodic follow-up
- Provocative tests rarely used today

Primary Angle-Closure (PAC)

- **Gonioscopy:** ITC >180° **with** IOP ↑ and/or PAS
- **Symptoms:**
  - **Subacute PAC**: intermittent attacks
    • transient blurring, colored haloes, brow/eye pain
    • episodes: 40–50 mmHg for minutes–hours
  - Triggered by:
    - **Dim light watching TV**, dark room, reading
    - **Pharmacological mydriasis**
    - Prone position
- **Between attacks:** asymptomatic, white eye

Treatment

- **Laser Peripheral Iridotomy (LPI)** is treatment of choice
- Stop progression → prevent acute attacks

Acute Primary Angle Closure (APAC)

(Previously: *Acute Congestive Glaucoma*)

Symptoms

- Markedly reduced vision (6/60 to HM) – *d/t epithelial edema*
- Severe ocular/periocular pain, headache – *d/t rapid increase in IOP*
- Nausea/vomiting, abdominal discomfort
- **Colored haloes & "smoke-filled room" vision**

Signs

- **IOP 50–80 mmHg**
- Conjunctival hyperemia + **violaceous** ciliary flush
- **Corneal epithelial edema**
- **Mid-dilated, vertically oval, non-reactive pupil –** *d/t iris spincter ischemia & paresis by increased IOP*
- **Shallow AC** + flare
- Fellow eye: **occludable angle**

Late findings (after resolution)

- Iris atrophy, **glaukomflecken**
- PAS formation, irregular pupil
- Optic nerve: pallor/cupping depending on damage

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- ***Vogt's Triad: Glaukomflecken, Iris atrophy patch & middilated/non reacting pupil***

INVESTIGATIONS

- **Gonioscopy** — essential for diagnosis
- **AS-OCT / UBM** — identify mechanism (pupillary block vs plateau iris)
- **AC depth measurement** — Van Herick, biometry if considering lens removal
- **Posterior segment B-scan** — if secondary causes suspected
- **Provocative tests** (rare): ***Dark-room test, Mydriatic test, Prone Test, Phenylephrine-Pilocarpine test, Triple Test*****-------** *IOP raise >8mmHg significant*
- **VFD in ACG:** Generalized field constriction

DIFFERENTIAL DIAGNOSIS OF ACUTE IOP RISE

- Lens-induced angle closure (swollen/subluxated lens)
- Malignant glaucoma (aqueous misdirection)
- Neovascular glaucoma
- Hypertensive uveitis (HSV/CMV), Posner-Schlossman
- Scleritis with/without angle closure
- Pigment dispersion, PXF
- Orbital mass / retrobulbar hemorrhage
- Carotid-cavernous fistula

TREATMENT

A) APAC — Emergency Management

Initial

- Patient **supine**
- **Acetazolamide 500 mg** IV/PO (avoid in sulfa allergy, topiramate-induced cases)
- Topicals:
  - **Timolol 0.5%**
  - **Apraclonidine 0.5–1%**
  - **Prednisolone 1%**
  - **Pilocarpine** (only if IOP <40 mmHg)

Resistant cases

- Indentation technique to force aqueous into angle
- Hyperosmotics:
  - **Mannitol 20%** 1–2 g/kg IV
  - Oral glycerol/isorbide
- Early **Laser PI / iridoplasty** after clearing cornea
- **Paracentesis** if needed

Definitive

- **Bilateral Laser Iridotomy** after breaking attack
- Continue topical steroids, aqueous suppressants

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- Gonioscopy MUST confirm angle opening

If persistent IOP ↑

- **Lens extraction** (especially if phacomorphic)
- Trabeculectomy (risk of malignant glaucoma)
- Cyclodiode laser in refractory cases

B) PACS / PAC / PACG Long-Term Management

- **Laser PI** — first-line for majority
- **Medical therapy** similar to POAG if IOP remains high
- If inadequate angle widening:
  - **Lens extraction** superior — EAGLE Study evidence
    → better IOP control & cost-effective when:
    - IOP >29 mmHg **or**
    - Established PACG

Drugs Causing Acute Angle-Closure Glaucoma (AACG)

1. Anticholinergics

- Atropine
- Tropicamide
- Scopolamine

2. Cholinergics

- Pilocarpine *(may precipitate angle closure in special situations such as lens-induced block or ciliary block)*

3. Adrenergic Agonists

- Phenylephrine
- Pseudoephedrine

4. Sulfonamide-related Drugs

- Topiramate *(most important)*
- Acetazolamide *(rare paradoxical reaction)*

(Topiramate-Induced Angle Closure

- Mechanism: Ciliochoroidal effusion → Anterior rotation of the ciliary body → Forward displacement of the lens–iris diaphragm → Acute myopia + secondary angle-closure glaucoma.
- IOP-lowering drugs: Use aqueous suppressants (hypotensive agents).
- Avoid: Miotics (e.g., pilocarpine) *(worsen anterior displacement)*.
- Treatment: Stop topiramate immediately + cycloplegics (± topical steroids if indicated).)

5. Antidepressants

- Amitriptyline
- Fluoxetine

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Causes of Colored Halos

1. Conjunctivitis *(mucus on cornea)*
2. Incipient cataract
3. Vitreous opacities
4. Snow blindness
5. Fog
6. Acute congestive glaucoma *(corneal epithelial edema)*

Fincham (Stenopaic Slit) Test

Purpose: Differentiates halos of incipient cataract from acute congestive glaucoma.

Condition	Result
Acute congestive glaucoma	Halo remains intact
Incipient cataract	Halo breaks into component colors

Differential Diagnosis of Acute Congestive Glaucoma

- Ciliary block (malignant) glaucoma
- Neovascular glaucoma (NVG)
- Iridocorneal Endothelial (ICE) syndrome
- Plateau iris syndrome
- Phacomorphic glaucoma

Signs of Previous Attack of Angle Closure

- Iris pigment on corneal endothelium *(endothelial dusting/Krukenberg pigment)*
- Peripheral anterior synechiae (PAS)
- Sectoral iris atrophy
- Posterior synechiae (PS)
- Mid-dilated sluggish pupil
- **Glaukomflecken** *(anterior subcapsular lens opacities)*
- Visual field defects (VFD)
- Optic disc cupping

Q. Causes of PAS: PACG, Uveitis, ICE syndrome, Trauma, Secondary Glaucoma

Q. Inverse Glaucoma : Microspherophakia → Miotics → Ciliary muscle contraction → Zonules slacken → ↑ Lens–iris contact → Forward displacement of lens → Inverse pupillary block → Secondary angle-closure glaucoma

Q. Indications for Trabeculectomy in PACS:

- Progressive Optic nerve head damage
- PAS > 270°

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- Failure to achieve Target IOP

Absolute Glaucoma

End-stage glaucoma presenting as a painful blind eye (NPL) with very high IOP.

- Painful blind eye
- Corneal & conjunctival congestion (CCC)
- Shallow anterior chamber
- Atrophic iris
- Band-shaped keratopathy (BSK)
- Increased (very high) IOP
- Optic atrophy

Treatment *(Goal: Pain Relief)*

- Topical antiglaucoma medications (AGMs)
- Topical steroids
- Cycloplegics
- Cyclocryotherapy / Cyclodestructive procedures
- Retrobulbar alcohol injection
- Evisceration (refractory painful blind eye)

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CLASSIFICATION OF SECONDARY GLAUCOMA

Secondary glaucoma = **elevated IOP due to an identifiable ocular/systemic cause** with structural or functional damage to aqueous outflow pathways.

CLASSIFICATION BASED ON MECHANISM OF IOP ELEVATION

A. SECONDARY OPEN-ANGLE GLAUCOMA (SOAG)

Definition:

Angle open on gonioscopy; obstruction to aqueous outflow occurs at **pre-trabecular, trabecular, or post-trabecular level**.

1. Pre-Trabecular Obstruction

(*Membrane covering trabecular meshwork*)

Physical membrane blocks aqueous access to TM.

Causes

Fibrovascular membrane

- **Neovascular glaucoma**
  - Due to retinal ischemia (PDR, CRVO)
  - Fibrovascular membrane over angle → progressive PAS

Endothelial membrane

- **ICE syndrome** (Iridocorneal Endothelial syndrome)
- Posterior polymorphous dystrophy

Epithelial membrane

- Epithelial downgrowth (post-surgery/trauma)

Connective tissue membrane

- Post-penetrating trauma

Inflammatory membrane

- Chronic uveitis

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2. Trabecular Obstruction

(*TM clogged or structurally altered*)

A. Clogging of Trabecular Meshwork

Cause	Obstructing Material
Pigmentary glaucoma	Pigment granules
Red cell glaucoma	Fresh RBCs
Ghost cell glaucoma	Degenerated RBCs
Phacolytic glaucoma	Macrophages + lens proteins
Pseudoexfoliation glaucoma	PXF material
Hypertensive uveitis	Inflammatory cells + proteins

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Cause	Obstructing Material
Intraocular hemorrhage	RBC debris
Tumors	Neoplastic cells
Post-surgical	Viscoelastic
Aphakia	Vitreous in angle

B. Trabecular Meshwork Dysfunction / Structural Alteration

- Steroid-induced glaucoma
- Uveitis (trabeculitis)
- Scleritis
- Chemical burns
- Toxic reactions
- Trauma (angle recession → TM scarring)
- Post-radiation

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3. Post-Trabecular Obstruction

(*Distal to TM — elevated episcleral venous pressure or Schlemm canal pathology*)

A. Elevated Episcleral Venous Pressure (EVP)

- Carotid–cavernous fistula
- Cavernous sinus thrombosis
- Sturge–Weber syndrome
- Superior vena cava obstruction
- Retrobulbar tumors
- Thyroid ophthalmopathy

B. Schlemm Canal Abnormalities

- Collapse/absence
- Synechial obstruction
- IOL/iris–vitreous block

B. SECONDARY ANGLE-CLOSURE GLAUCOMA (SACG)

Angle physically closed by peripheral iris apposition or synechiae.

With Pupillary Block

(*Relative block at pupil → iris bombe → angle closure*)

Causes

- 360° posterior synechiae (Seclusio pupillae) — recurrent uveitis
- Phacomorphic glaucoma (intumescent cataract)
- Subluxated/dislocated lens
- Aphakic pupillary block
- Anterior chamber IOL without patent iridotomy
- Capsular block syndrome (post-phaco, iris–capsule apposition)

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Without Pupillary Block

A. Anterior Pulling Mechanism

(*Membrane contraction pulling iris over angle*)

- Neovascular glaucoma (fibrovascular PAS)
- ICE syndrome
- Chronic uveitis (PAS)
- Post-trauma inflammatory contraction

B. Posterior Pushing Mechanism

(*Pressure from behind iris*)

- Cilio-choroidal effusion
- Malignant glaucoma (ciliolenticular block)
- Ciliary body/iris cyst
- Intraocular tumors
- Retrolenticular tissue contraction:
  - PVR
  - ROP
  - PHPV
- Capsular block syndrome (without iris–capsule contact)

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Epithelial Ingrowth

- Rare complication after anterior segment surgery/trauma.
- Caused by migration and proliferation of corneal/conjunctival epithelium into the anterior chamber.

Mechanism of IOP Rise

- Trabecular obstruction by:
  - Epithelial membrane
  - Secondary synechial angle closure
  - Shed epithelial + inflammatory cells
    → leads to difficult-to-control glaucoma.

Clinical Features / Diagnosis

- Persistent postoperative anterior uveitis
- Greyish translucent membrane with *scalloped border* on posterior cornea at wound site
- Cystic or fibrous forms (better prognosis)
- Pupillary distortion

Treatment — Goal: Eradicate epithelium

1. **Block excision**
   - Remove involved corneoscleral tissue + adjacent iris + pars plicata
   - Repair with tectonic corneoscleral graft
   - Argon laser may delineate affected area
2. **Cryotherapy**
   - Destroys residual epithelium on posterior cornea, angle, ciliary body
   - Air bubble protects other structures
3. **Chemotherapy**
   - Intracameral 5-FU (variable benefit)
   - Intravitreal methotrexate (400 µg/0.1 ml q2 weeks × 6) — promising
4. **Glaucoma drainage devices**
   - For uncontrolled IOP when excision not possible

Prognosis

- Often poor due to difficulty fully eliminating epithelial cells.

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Pigmentary Glaucoma

Definitions

- **PDS:** Dispersion of iris pigment onto corneal endothelium (Krukenberg spindle), iris transillumination defects, and heavy trabecular meshwork pigmentation.
- **Pigmentary Ocular Hypertension (POH):** PDS with elevated IOP but no glaucomatous optic neuropathy.
- **Pigmentary Glaucoma (PG):** PDS with glaucomatous optic neuropathy, may have elevated or normal IOP; secondary open-angle glaucoma.

Epidemiology & Risk Factors – *white, young, myopic, male*

- **Gender & Age:** More common in young myopic males (20–40 years).
- **Race:** Predominantly Caucasians; rare in Africans/Asians but more severe if affected.
- **Genetics:** Autosomal dominant inheritance (Ch 7q & 18q).
- **Other risk factors:** Myopia (80%), exercise, accommodation, frequent blinking, family history, presenting IOP >21 mmHg.

Pathogenesis

I. Mechanical Theory (Campbell's Theory)

- Posterior iris rubs against lens zonules → pigment release.
- Reverse pupillary block → posterior bowing (concavity) of iris → ↑ irido-zonular contact → ↑ pigment shedding.
- Pigment blocks trabecular meshwork → ↓ aqueous outflow → ↑ IOP (pigmentary glaucoma).
- Pigment release decreases with age; steroid responsiveness may be increased.

II. Inherited Defect Theory

- Inherited abnormality of the iris pigment epithelium (PE).
- Focal atrophy / hypopigmentation of iris PE.
- Delayed melanogenesis.
- Hypertrophy of iris dilator muscle.

III. Anderson et al. Theory

- Mutations in melanosomal protein genes → abnormal pigment production/release → Pigment Dispersion Syndrome (PDS) → Pigmentary Glaucoma (PG).

Clinical Features

(Typical History: Young myopic male c/o: blurry vision, headache & N/V after exercise—release of pigments—increased IOP—Corneal Edema)

- **Stages:**
    1. Inactive pigment dispersion, stable IOP
    2. Active pigment dispersion, stable IOP
    3. Active pigment dispersion, progressive glaucoma

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4. Inactive pigment dispersion, progressive glaucoma

- **Anterior segment:**
  - **Cornea:** Krukenberg spindle, Endothelium pleomorphism & polymegathism
  - **Iris:** Radial mid-peripheral transillumination defects, pupillary ruff loss, heterochromia in dark irides, anisocoria, pigment granules
  - **Anterior chamber:** Floating pigment granules, deep AC
  - **Lens:** Pigment on anterior lens surface, Zonules , Scheie stripe on Equator & Zentmayer lines on posterior lens surface
- **Gonioscopy:** Wide open-angle, mid-peripheral iris concavity, homogeneous trabecular meshwork pigmentation, pigment along Schwalbe's line (Sampolesi's line)
- **Posterior segment:** Peripheral retinal pigmentation, lattice degeneration, increased risk of retinal detachment, glaucomatous optic neuropathy (may be asymmetrical)

Investigations

- **UBM / AS-OCT:** Confirm iris concavity and irido-zonular contact
- **IOP monitoring:** Volatile, can fluctuate widely
- **Optic nerve & visual fields:** Assess glaucomatous damage
- **Differential diagnosis:** Pseudoexfoliation syndrome, uveitis, iris/ciliary body cysts, melanoma

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Management

Monitoring: Annual review for PDS; 4–6-monthly for established PG.
Lifestyle: Avoid vigorous exercise that may trigger pigment release.
Medical Therapy – *Pilocarpine 1%, CAI (Dorzox), B-blocker (timolol)*

- Similar to POAG (beta-blockers, prostaglandin analogues)
- **Miotics:** Reduce irido-zonular contact, facilitate outflow, poorly tolerated in young patients; risk of inducing retinal detachment
- **Thymoxamine:** Induces miosis without accommodation spasm

Laser Therapy

- **Laser trabeculoplasty:** Effective, especially in young patients; treat cautiously with heavily pigmented angles (ALT,SLT)
- **Laser peripheral iridotomy (LPI):** Prophylactic in young myopic males to reverse iris concavity, prevent reverse pupillary block; does not reduce glaucoma risk

Surgery

- **Trabeculectomy:** Indicated in uncontrolled PG; success similar to POAG but with risk of hypotony and suprachoroidal hemorrhage
- **Adjunctive antimetabolites** improve outcomes in younger patients

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❖ **DD of Pigments at Angle:** Aging, Pigmentary glaucoma (PG), Pseudoexfoliation syndrome (PXF), Post-trauma, Post-surgery, Uveitis, Previous acute angle-closure (ACG) attack, IOL in sulcus, Ocular melanosis, Iris cysts.

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- **DD of Pigmentary Glaucoma (PG):** Pseudoexfoliation (PXF) deposits, Angle recession glaucoma, Traumatic ocular melanosis, Post-surgery/Post-laser pigment dispersion, Herpes zoster (HZ), Iris & ciliary body cysts, Pigmented trabecular tumor, Siderosis.

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Pseudoexfoliation Glaucoma

Introduction

- Grey white fibrillogranular ECM material surrounded by GAG
- PXF is a systemic disorder with deposition of **whitish, dandruff-like material** on anterior segment structures (lens, iris, trabeculum) and sometimes in skin, heart, lungs, kidneys, and meninges.
- **PXG**: Secondary open-angle glaucoma due to trabecular obstruction by PXF material.
- Prevalence rises with age; rare before 50, up to 5% at 75–85 years.
- More common in **women**, highest prevalence in **Scandinavians**, but occurs worldwide.
- At diagnosis, **15–30%** of PXS patients have glaucoma; cumulative 5-year risk for treatment ~60%.

Demography & Genetics

- **Age:** PXF develops after ~40 years; PXG mostly after 60 years.
- **Sex:** PXF more common in females; glaucoma more common in males.
- **Race:** Highest prevalence in Caucasians; occurs in all races.
- **Laterality:** Usually asymmetric; one eye may be more affected.
- **Genetics:** Strong association with **LOXL1 gene SNPs** (rs2165241, rs1048661, rs3825942).

Pathogenesis: *Microfibril Theory, BM Theory, GAG Theory*

- Fibrillogranular material composed of abnormal elastic microfibrils, basement membrane material, and glycosaminoglycans.
- Secreted by pre-equatorial lens epithelium (abnormal BM of aging epithelial cell) → deposited on lens capsule, zonules, iris, trabeculum.
- **Mechanism of glaucoma:**
    - Secondary open-angle: Trabecular obstruction by PXF material and liberated iris pigment.
    - Rarely, secondary chronic angle-closure due to weak zonules → lens–iris diaphragm movement + PAS formation.
- Systemic associations: High-tone hearing loss, cardiovascular disorders.
- Risk factors for PXG: Older age, male sex, higher baseline IOP, zonular laxity.

Clinical Features

PXF Material

- **Cornea:** Dandruff-like deposits on endothelium; sometimes Krukenberg spindle; polymegathism, pleomorphism, decreased endothelial count
- **Anterior chamber:** Mild aqueous flare due to pseudouveitis.
- **Iris:**
    - Pupillary border deposits

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- Patchy peripapillary transillumination defects ("**moth-eaten**")
- Sphincter atrophy, pigment dispersion, intrastromal hemorrhages
- Poor mydriasis; posterior synechiae may develop

- **Lens:**
  - Central disc, clear intermediate zone (*d/t iris movement*), peripheral granular band—**Target Sign**
  - Zonular instability → phacodonesis, risk during surgery (*PXF—proteolytic enzymes—zonular disintegration)*
  - Associated nuclear cataract

Anterior Chamber Angle

- Patchy trabecular and Schwalbe line hyperpigmentation; Sampaolesi line may be present.
- Increased risk of angle closure due to zonular laxity.

IOP & Glaucoma

- Often **unilateral initially**, may rise acutely or chronically.
- PXG progresses faster than POAG; poorer response to medications; higher risk of visual loss.

Investigations

- **Gonioscopy:** Trabecular hyperpigmentation, Sampaolesi line.
- **Anterior segment OCT / UBM:** Assess angle and zonular stability.
- **IOP monitoring:** Chronic fluctuations; may mimic acute angle closure.
- **Other:** Assess optic nerve, visual fields.

Management

Medical

- Similar to POAG; higher failure rate.
- **Miotics (pilocarpine):** Reduce pupil movement and iris-lens contact.

Laser

- **SLT / ALT:** Effective initially; >50% failure by 5 years.
- **Laser peripheral iridotomy:** Rarely used; may reduce iris-lens contact.

Surgical

- **Trabeculectomy / Filtration surgery:** Similar success as POAG.
- **Phacoemulsification:** Can lower IOP; higher complication risk due to poor mydriasis, weak zonules, endothelial compromise.
- **Management of PXF Cataract:** Visco elastics—Chondroitin sulfate (endo friendly), Iris Hook/ Kuglen hook, CTR, Heparinized IOL
- **Trabecular aspiration:** Short-term benefit; may be combined with other procedures.

Prognosis

- Worse than POAG due to higher, fluctuating IOP.

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- Rapid progression, especially in unilateral cases; careful follow-up (≤6 months) recommended.
- Zonular weakness increases surgical complexity and complication risk.

Intraocular Complications of PXF

- **Trabecular meshwork (TM):** POAG, ACG
- **Lens/Zonules:** Phacodonesis, Lens subluxation, Nuclear cataract, Zonular dialysis
- **Iris:** Posterior synechiae (PS), Poor mydriasis (miosis), Sphincter degeneration
- **Cornea:** Endothelial decompensation
- **Retina:** Central retinal vein occlusion (CRVO), Retinal detachment (RD)

True Exfoliation (Glassblower's Cataract)

- Delamination (splitting) of the anterior lens capsule due to chronic infrared (IR) exposure.
- Classically seen in: Glassblowers.
- Pathology: Separation of the superficial from the deeper layer of the anterior lens capsule.
- Genetics: LOXL1 gene (exon 1 polymorphism) association.

DDx of Pseudoexfoliation (PXF)

- True exfoliation syndrome (capsular delamination)
- Uveitis
- Amyloidosis
- Pigment Dispersion Syndrome (PDS)
- Pigmentary Glaucoma (PG)
- Ocular melanosis
- Ocular melanoma (uveal melanoma)

Phacomorphic Glaucoma	PACG
Unilateral (U/L)	Usually bilateral (B/L)
Intumescent (swollen) cataract	Normal lens
Asymmetrically shallow anterior chamber	Bilaterally shallow anterior chambers
Fellow eye usually normal	Fellow eye also has narrow angle
Treatment: Cataract extraction	Treatment: Laser Peripheral Iridotomy (LPI)

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PXF	PDS / PG
Older age (>50 years)	Younger age (20–40 years)
M = F	M > F
Worsens with age	Improves in later life
Open angle (OA) ± narrow angle/angle closure (AC)	Open angle (OA)
Deposition of flaky pseudoexfoliative material	Pigment released from posterior iris
Poor mydriasis	Krukenberg spindle, iris transillumination
Weak zonules (phacodonesis)	Deep anterior chamber
Peripupillary iris transillumination	Mid-peripheral iris transillumination
Sampaolesi's line (anterior to Schwalbe's line)	Dense, uniform trabecular meshwork pigmentation
Management: Primarily surgical	Management: Primarily medical
40% progress to pseudoexfoliative (capsular) glaucoma	35% of PDS progress to pigmentary glaucoma

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Lens-Induced Glaucoma

Lens-induced glaucomas are secondary glaucomas caused by **mechanical or biochemical effects of the crystalline lens**, either through obstruction of the trabecular meshwork or pupillary block.

Classification

I. Lens-Induced Secondary Angle-Closure Glaucoma

1. **Phacomorphic glaucoma** – due to swollen (intumescent) cataract
2. **Phacotopic glaucoma** – due to anterior lens displacement
3. **Pupillary block from lens subluxation/dislocation/ Ectopia Lentis**
4. **Pupillary block from microspherophakia**

II. Lens-Induced Secondary Open-Angle Glaucoma

1. **Phacolytic glaucoma** – leakage of lens proteins through intact capsule
2. **Lens particle glaucoma** – obstruction by lens fragments after trauma or surgery
3. **Phacoantigenic (phacoanaphylactic) glaucoma** – immune reaction to lens proteins

---

1. Phacomorphic Glaucoma (Angle-Closure)

- **Pathogenesis:** Acute angle-closure caused by **intumescent cataract**. Lens enlargement pushes iris forward, causing pupillary block and iris bombe. Equatorial lens growth stretches zonules; anteroposterior growth increases iridolenticular contact.
- **Clinical features:** Acute congestive glaucoma, shallow anterior chamber, mid-dilated pupil, corneal edema, cataractous lens. Fellow eye may have normal AC.
- **Investigations:** Anterior segment OCT or UBM to evaluate AC depth and lens position.
- **Management:**
    - **Medical:** IOP control with systemic acetazolamide, IV mannitol, topical beta-blockers, steroids. Avoid miotics (may worsen block).
    - **Laser:** PI may help but sometimes ineffective; iridoplasty can be temporizing.
    - **Surgical:** Cataract extraction with PCIOL once IOP controlled and eye quiet.

---

2. Lens Subluxation / Dislocation Glaucoma (Pupillary Block)

- **Causes:**
    - Congenital (ectopia lentis, Marfan's, Weill-Marchesani, homocystinuria)
    - Traumatic (closed globe injury)
    - Spontaneous / secondary (PXF, hypermature cataract, buphthalmos, high myopia, uveitis)
- **Mechanism:** Pupillary block due to lens dislocated into AC or tilted against iris; vitreous herniation; may lead to chronic angle closure with PAS.
- **Management:**

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- Initial IOP reduction: Osmotic agents, cycloplegics.
- Laser PI or iridoplasty may help bypass pupillary block.
- Definitive: Surgical lens removal; AC, iris-, or scleral-fixated IOL if needed.

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3. Pupillary Block Glaucoma from Microspherophakia

- **Mechanism:** Globular lens causes pupillary block and angle-closure.
- **Management:** Cycloplegics to dilate pupil, tighten zonules, pull lens posteriorly. Miotics contraindicated ("inverse/paradoxical glaucoma").

---

4. Phacolytic Glaucoma (Open-Angle)

- **Pathogenesis:** Secondary open-angle glaucoma caused by leakage of **high molecular-weight lens proteins** from a **hypermature cataract**. Proteins obstruct trabecular meshwork; macrophages contribute.
- **Clinical features:**
  - Pain, poor vision from cataract
  - Deep AC, corneal edema
  - White proteinaceous particles in AC; may form pseudohypopyon
  - Gonioscopy: open angle with lens material
- **Management:**
  - Medical: IOP control
  - Surgical: Cataract extraction with PCIOL after controlling inflammation
- **Phacolytic glaucoma from posteriorly dislocated lens:**
  - Lens degenerates in vitreous → protein release → trabecular obstruction
  - Management: Pars plana lensectomy with vitrectomy

Hypermature cataract
↓
Leakage of high-molecular-weight lens proteins through an intact but permeable lens capsule
↓
Macrophages engulf lens proteins
↓
Protein-laden macrophages + lens proteins obstruct trabecular meshwork
↓
↓ Aqueous outflow
↓
↑ IOP
↓
Secondary Open-Angle Glaucoma (Phacolytic glaucoma)

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5. Lens Particle Glaucoma (Open-Angle)

- **Pathogenesis:** Open-angle glaucoma caused by lens fragments in AC after trauma or surgery/laser. Inflammation contributes.
- **Clinical features:** Acute IOP rise, lens particles visible in AC.
- **Management:**
  - Medical IOP control
  - Irrigation–aspiration of lens particles
  - Cycloplegics and topical steroids

---

6. Phacoantigenic (Phacoanaphylactic) Glaucoma – *granulomatous reaction*

- **Pathogenesis:** Immune reaction to lens proteins released after surgery, trauma, or capsule rupture. Trabecular meshwork clogged by inflammatory cells + lens matter.
- **Clinical features:** Fulminant inflammation, KPs on endothelium, low-grade vitritis, synechiae, residual lens material.
- **Management:**
  - Medical: IOP control
  - Steroids + cycloplegics for inflammation
  - Irrigation–aspiration of lens material after controlling inflammation

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Inflammatory Glaucoma

Classification of Inflammatory Glaucomas

I. Non-specific inflammatory glaucomas

1. **Open-angle inflammatory glaucoma**
    - Acute
    - Chronic
2. **Angle-closure inflammatory glaucoma**

II. Specific hypertensive uveitis syndromes

1. **Fuchs Heterochromic Uveitis Syndrome (FUS)**
2. **Posner-Schlossman Syndrome (Glaucomatocyclitic crisis)**

I. Open-Angle Inflammatory Glaucoma

Acute

- **Mechanisms:**
    - Trabecular clogging by inflammatory cells, exudates, or turbid aqueous
    - Trabecular edema due to trabeculitis
    - Prostaglandin-induced rise in IOP
- **Common associations:** Herpes zoster iridocyclitis, HSV, keratouveitis, toxoplasmosis, pars planitis, rheumatoid arthritis
- **Clinical features:**
    - Signs of acute iridocyclitis with raised IOP
    - Open angle on gonioscopy
    - IOP usually normalizes after acute inflammation subsides
- **Management:**
    - Treat iridocyclitis (topical steroids + cycloplegics)
    - Control IOP: hyperosmotic agents, oral acetazolamide, topical beta-blockers or CAIs
    - **Avoid:** pilocarpine, prostaglandin analogues

Chronic

- **Mechanisms:** Chronic trabeculitis, trabecular scarring
- **Clinical features:**
    - Persistently raised IOP
    - Open angle, signs of prior uveitis
    - Possible glaucomatous optic neuropathy
- **Management:**
    - Topical beta-blockers, CAIs, alpha-agonists
    - Trabeculectomy with steroids if medical therapy fails
    - Cyclodestructive procedures if surgical options fail
- **Special note:**

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- **Grant's syndrome:** Chronic open-angle glaucoma with minimal inflammation; responds well to topical steroids.

II. Angle-Closure Inflammatory Glaucoma

1. With Pupillary Block

- **Mechanism:** 360° posterior synechiae (seclusio pupillae) → iris bombe → appositional angle closure → PAS if untreated
- **Clinical features:** Raised IOP, shallow AC, iris bombe, seclusio pupillae
- **Management:**
  - Prophylaxis: aggressive treatment of acute uveitis with topical steroids + atropine
  - Curative: IOP-lowering medications (avoid miotics), laser or surgical iridotomy

2. Without Pupillary Block

- **Mechanism:** Organization of inflammatory debris in the angle → iris pulled over trabeculum → progressive synechial angle closure
- **Risk factors:** Granulomatous inflammation, pre-existing narrow angles, rubeosis iridis
- **Clinical features:** Raised IOP, shallow AC, PAS formation, signs of prior inflammation
- **Management:**
  - Medical: beta-blockers, CAIs, alpha agonists
  - Surgical: trabeculectomy with mitomycin C or tube implants if medical therapy fails

---

III. Specific Hypertensive Uveitis Syndromes

A. Fuchs Heterochromic Uveitis Syndrome (FUS)

Symptoms:

- Gradual blurring of vision (most commonly due to posterior subcapsular cataract [PSC])
- Floaters
- Difference in eye color (heterochromia)

Signs:

1. **Cornea:**
   - Fine stellate keratic precipitates (KPs) scattered over endothelium, sometimes pathognomonic
2. **Anterior Chamber:**
   - Mild aqueous flare
   - Cells ≤ +2
3. **Iris:**
   - Heterochromia (common)
   - Diffuse stromal atrophy → "moth-eaten" appearance
   - Patchy posterior pigment atrophy (retroillumination defects)

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- Iris nodules along pupillary border
- Russell bodies (refractile iris crystals)
- Rubeosis iridis; recurrent hyphema possible (Amsler's sign)
- Posterior synechiae absent

4. **Lens:**
   - Posterior subcapsular cataract (common; often presenting symptom)
5. **Glaucoma (≈30% cases):**
   - Secondary open-angle, likely due to trabecular sclerosis; may be exacerbated by steroids
   - IOP: initially intermittent, later constant; sometimes triggered by cataract surgery
   - Gonioscopy: open-angle ± twig-like neovascularization

Treatment

- **Uveitis:** Steroid-resistant; steroids mainly for comfort
  - Posterior subtenon triamcinolone for floaters
  - Mydriatics usually unnecessary
- **Glaucoma:** Managed like POAG
  - Avoid pilocarpine and prostaglandin analogues
  - Medical therapy often fails (≈73%)
  - Surgery (trabeculectomy with antimetabolites or drainage devices) is mainstay

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B. Posner-Schlossman Syndrome (PSS) / Glaucomatocyclitic Crisis

- recurrent **unilateral acute IOP spikes**, rarely simultaneous in both eyes.

Etiology:

- Acute trabeculitis, possibly due to **HSV, CMV, H. pylori**
- **Prostaglandin E** elevation correlates with IOP rise
- Mostly **young adult males**; ~40% HLA-BW54 positive

Clinical Features:

- **Symptoms:** Mild discomfort, halos, blurred vision, minimal redness/pain
- **Signs:**
  - White eye (no congestion)
  - IOP 40–50 mmHg during attacks, normal between
  - Corneal edema, fine KPs after 2–3 days
  - Few AC cells, minimal flare, no posterior synechiae
  - Open-angle on gonioscopy, no PAS
- **Course:**
  - Usually unilateral; ~50% occasional bilateral attacks
  - Intervals lengthen over time
  - Recurrent attacks → chronic open-angle glaucoma

Investigations:

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- PCR for CMV/HSV/VZV in aqueous
- OCTA, laser Doppler flowmetry for optic nerve perfusion

Treatment:

- **Uveitis:** Topical steroids, oral NSAIDs; antiviral therapy for CMV (ganciclovir drops/oral/IV)
- **Glaucoma:** Topical agents (beta-blockers, alpha-2 agonists, CAIs), oral acetazolamide if needed

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Key Principles

- Control **inflammation first**, then IOP
- Avoid **miotics and prostaglandin analogues** in uveitic eyes
- Preoperative **control of inflammation** is crucial before surgery
- Steroid-induced IOP elevation should be differentiated from inflammatory glaucoma

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Gonioscopy Findings After Ocular Trauma

- Angle recession
- Trabecular meshwork tears
- Iridodialysis
- Cyclodialysis
- Intraocular foreign body (IOFB)
- Peripheral anterior synechiae (PAS)
- Angle hematoma (blood in the angle)

Cyclodialysis: Disinsertion of the ciliary body from the scleral spur (usually due to blunt trauma).

Cyclodialysis cleft
↓
Communication between anterior chamber (AC) & suprachoroidal space
↓
↑ Uveoscleral outflow
↓
Ocular hypotony (↓ IOP)

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Angle Recession Glaucoma

Introduction

- ARG is a **secondary open-angle glaucoma** resulting from **blunt ocular trauma**.
- **Angle recession:** tear of the ciliary body face between iris root and scleral spur.
- Often follows **traumatic hyphema**; IOP elevation arises from **trabecular meshwork fibrosis**, not the recession itself.
- Glaucoma develops in a **small percentage (<10%)** over years; cases reported even **50 years post-injury**.
- Fellow eye risk: ~50% higher than the affected eye.
- Usually **unilateral**, trauma history may be forgotten.

Etiology & Risk Factors

- Occurs in up to 60% of **non-penetrating ocular trauma**
- Strongly associated with **traumatic hyphema** (60–100% incidence)
- Common causes: recreational injuries, assault; sometimes no recalled trauma
- Risk factors for chronic glaucoma:
  - Angle recession >180°
  - Hyphema
  - Lens displacement
  - Elevated baseline IOP
  - Angle pigmentation

Pathophysiology

- Blunt trauma → aqueous pushed laterally/posteriorly → traction on iris root → tear between **longitudinal & circular ciliary body fibers**
- Ciliary artery rupture → hyphema → acute IOP spike
- Chronic fibrosis of **trabecular meshwork & Schlemm's canal** → delayed IOP rise
- Additional mechanisms:
  - Loss of ciliary muscle tension on scleral spur → Schlemm's canal narrowing
  - Hyaline membrane formation across trabecular meshwork → impaired outflow

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Clinical Features

Symptoms:

- Painless, progressive vision loss

Signs:

- **Slit-lamp:** iris sphincter tears, corneal scars, Vossius ring, iridodialysis, traumatic cataract, phacodenesis, hyphema
- **Gonioscopy:**
  - Widened ciliary body (CB) band
  - Absent/torn iris processes
  - Glistening white scleral spur—*d/t tearing of uveal meshwork from surface*

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  - Irregular trabecular pigmentation
  - PAS at either end of recession
  - Chronic cases: fibrosis & hyperpigmentation
- Post-trauma gonioscopic view often shows widening of inferior CB band and irregular pigmentation

Classification of Angle Recession:

- Shallow: increased visibility of CB & scleral spur
- Moderate: tear between longitudinal & circular fibers
- Deep: irregular widening of CB band

Glaucoma Development:

- May occur **months to years later**
- Risk correlates with **clock-hour extent** of recession (>180° higher risk)
- Fellow eye may develop POAG in ~50% of patients

Diagnosis

- Gonioscopy: widened CB band with angle changes
- IOP elevation and optic nerve damage confirm ARG
- Slit-lamp findings support prior trauma
- Annual gonioscopy recommended in patients with hyphema or significant blunt trauma

Differential Diagnosis:

- Unilateral steroid-induced glaucoma
- Post-surgical glaucoma
- Uveitis–glaucoma–hyphema syndrome
- Iridocorneal endothelial syndrome
- Carotid-cavernous fistula
- Other asymmetric glaucomas: pseudoexfoliation, pigmentary glaucoma

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Management

Medical Therapy:

- Topical aqueous suppressants: beta-blockers, CAIs, alpha-agonists
- Avoid prostaglandin analogues in acute trauma (use later)
- Avoid pilocarpine (may worsen angle recession)
- Cycloplegics if pupillary block due to lens dislocation

Laser Therapy:

- Argon or SLT generally **ineffective**; high failure rates

Surgical Therapy:

- Trabeculectomy with MMC: effective, but higher failure risk than other glaucomas
- Glaucoma drainage devices: for refractory cases
- Cyclodestructive procedures: for eyes with limited visual potential

Follow-up:

- IOP yearly for patients with >2 quadrants of recession
- Regular monitoring and patient counseling

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CHILDHOOD (PAEDIATRIC) GLAUCOMAS

Heterogeneous group of glaucomas occurring in **children**

- Characterized by **raised IOP and/or glaucomatous optic neuropathy**
- Due to **elastic sclera and cornea**, raised IOP causes:
  - Buphthalmos
  - Increased corneal diameter
  - Axial elongation
- **CGRN classification** is the current standard

CGRN DEFINITION OF CHILDHOOD GLAUCOMA

Diagnosis: ≥2 criteria required

1. **IOP > 21 mm Hg** *(Interpret cautiously under anaesthesia)*
2. **Optic disc changes**
   - Progressive ↑ CDR
   - CDR asymmetry ≥ 0.2
   - Focal neuroretinal rim thinning
3. **Corneal changes**
   - Haab's striae:

     Corneal diameter: ≥11 mm (newborn), 12 mm (<1 year), 13 mm (any age)
4. **Progressive myopia / axial length increase**
5. **Reproducible glaucomatous visual field defect** (if testable)

GLAUCOMA SUSPECT (CGRN)

No IOP-related damage + ≥1 of the following

- IOP >21 mm Hg (on 2 occasions)
- Suspicious optic disc
- Suspicious visual field
- Increased corneal diameter or axial length with normal IOP

Reversible Cupping: Reversal of optic disc cupping due to elastic recoil of the lamina cribrosa and scleral canal after IOP normalization.

Occurs in: Young age, Short duration of raised IOP, Minimal retinal ganglion cell (RGC)/axonal loss

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CGRN CLASSIFICATION

A. PRIMARY CHILDHOOD GLAUCOMA *(No associated anomalies)*

I. Primary Congenital Glaucoma (PCG)

- **Neonatal:** 0–1 month
- **Infantile:** >1–24 months
- **Late-onset / late-recognized:** >2 years
- **Spontaneously arrested PCG:** Normal IOP + typical signs

II. Juvenile Open-Angle Glaucoma (JOAG)

Juvenile Open-Angle Glaucoma (JOAG)

- Onset 3–40 years
- Open angles, No ocular enlargement
- Often autosomal dominant
- No ocular/systemic anomalies
- Clinically similar to POAG → treat per POAG principles
- Meets CGRN glaucoma criteria

B. SECONDARY CHILDHOOD GLAUCOMA

I. Non-acquired Ocular Anomalies

Iridodysgenesis

- Ectropion uveae
- Iris hypoplasia
- Aniridia
- Microphthalmos / microcornea

Iridocorneal dysgenesis

- Posterior embryotoxon
- Axenfeld–Rieger anomaly/syndrome
- Peters anomaly/syndrome

Other ocular anomalies

- PFV
- Nevus of Ota
- Ectopia lentis
- Ectopia lentis et pupillae

II. Non-acquired Systemic Anomalies

- **Chromosomal:** Down, Edwards, Trisomy 13–18, Turner
- **Ectopia lentis syndromes:** Marfan, Weill-Marchesani, Homocystinuria
- **Phacomatoses:** Sturge–Weber (~50%), NF-1 (~25%)
- **Metabolic:** Lowe, Hurler, Zellweger, Rubinstein–Taybi
- **Connective tissue:** Stickler, Klippel–Trénaunay

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- **Congenital rubella**

III. Acquired Conditions

- **Trauma:** Angle recession, hyphaema, ghost cell, lens debris
- **Tumours:** Retinoblastoma, JXG, leukaemia, iris RMS
- **Uveitic glaucoma:** Open-angle / angle-closure
- **Steroid-induced:** VKC
- **Neovascular:** Retinoblastoma, Coats, FEVR
- **Secondary angle closure:** ROP, ciliary body cysts, topiramate
- **Raised EVP:** AV fistula, orbital lesions
- **Infections:** Toxoplasmosis, herpes, endophthalmitis
- **Malignant glaucoma:** Post-ocular surgery

IV. Post-cataract Surgery Glaucoma

- Aphakic / pseudophakic
- Lens debris / uveitic block
- Pupillary block
- Chronic open-angle glaucoma

Examination Under Anesthesia (EUA) Sequence

1. Tonometry *(immediately after induction, before intubation)*
2. External examination
3. Anterior segment examination
4. Corneal diameter measurement
5. Koeppe gonioscopy
6. Fundus examination *(optic disc evaluation)*
7. Ultrasound pachymetry (CCT)
8. Ultrasound biometry *(Axial Length ± B-scan if indicated)*
9. Optic nerve head photography + Cycloplegic refraction *(after pupillary dilatation)*
10. Appropriate surgical procedure *(if indicated)*

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PRIMARY CHILDHOOD GLAUCOMA

due to developmental angle anomalies

PATHOGENESIS

- Arrested development of **neural crest-derived angle structures**
- Primary site of obstruction: **Trabecular meshwork**
- **Barkan's membrane theory – obsolete**

NORMAL DEVELOPMENT OF ANTERIOR CHAMBER ANGLE

- Neural crest mesenchyme → anterior segment

Layer	Structure
1st	Corneal endothelium → Descemet's membrane
2nd	Corneal stroma
3rd	Iris stroma + pupillary membrane

Shields' Concept (1983)

- 5th month: Closed AC cavity
- Iris inserts anterior to primordial TM
- 3rd trimester: Endothelial regression + posterior uveal shift
- TM & Schlemm's canal develop
- Angle maturation completes by **1 year**

ABNORMAL ANGLE DEVELOPMENT (CURRENT VIEW)

I. Isolated Trabeculodysgenesis

- Abnormal TM only
- No other anomalies
- Iris insertion: **Flat** (common), **Concave** (less common)

II. Iridotrabeculodysgenesis

- TM + iris anomalies
- Anterior stromal hypoplasia (Axenfeld, Rieger, Peters)
- Hyperplasia (Sturge–Weber)
- Anomalous iris vessels
- Structural iris holes

III. Corneotrabeculodysgenesis

- **Peripheral:** Axenfeld anomaly
- **Mid-peripheral:** Rieger anomaly
- **Central:** Peters anomaly
- **Microcornea:** Microphthalmos, PFV
- **Megalocornea:** Axenfeld / X-linked (≠ buphthalmos)

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PRIMARY CONGENITAL GLAUCOMA

Primary congenital glaucoma (PCG) is a developmental glaucoma due to **isolated maldevelopment of the trabecular meshwork (trabeculodysgenesis)**, **without any other ocular anomalies** causing raised IOP.

Classification (based on age of onset):

1. **True / Neonatal Congenital Glaucoma** – IOP raised **in utero to 1 month** (~25–40%)
2. **Infantile Glaucoma** – Onset **>1–24 months (or before 3 yrs)** (~55–65%)
3. **Juvenile Glaucoma** – Onset **3–16 (or 40) yrs** (least common)
4. **Late-recognised PCG** – Diagnosed after age 2 yrs
5. **Spontaneously arrested PCG** – Normal IOP but typical PCG signs

Buphthalmos / Hydrophthalmos

- Occurs when onset is **before 3 years**
- Enlarged globe due to scleral elasticity from retained aqueous

Epidemiology & Genetics

- Incidence: **1/10,000–18,000** live births (higher with **consanguinity**: e.g., **1/2500 Middle East**, **1/3300 India**)
- Accounts for **50–70%** of pediatric glaucomas
- **Bilateral in ~70%**, often asymmetric
- **Male predominance (~65%)**
- Mostly **sporadic**; **AR inheritance** in ~10%
- **Genetics**:
  Key gene: **CYP1B1**
  Loci: **GLC3A, GLC3B, GLC3C**
- **Recurrence risks**:
  ~5% if one parent affected or one affected sibling
  ~25% if both parents are carriers

Pathogenesis

- **Trabeculodysgenesis** → poor aqueous outflow → raised IOP
- Angle appears covered with smooth membrane-like tissue

Clinical Presentation

Usually noticed by parents or pediatrician.

Classic Symptom Triad

- **Epiphora (watering)**
- **Photophobia**
- **Blepharospasm**

Other symptoms:

- Rubbing of eyes

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- Enlarged cloudy eyes in infants
- Progressive myopia/vision loss in older children
- Rarely pain

Signs

Evaluation often requires **EUA** (Exam Under Anaesthesia)

Anterior Segment

1. **Corneal changes**
    - **Oedema** → first sign
    - **Corneal enlargement**
        - Normal infant cornea: **10.5 mm**
        - **>13 mm** diagnostic; **>16 mm → poor prognosis**
    - **Haab Striae** – curvilinear Descemet tears (glassy "railroad tracks")
    - **Increased thickness**, scarring, vascularization
2. **Scleral thinning** → blue hue
3. **Deep anterior chamber**
4. **Iris** – usually normal but may show **stromal hypoplasia, iridodonesis** late
5. **Lens** – stretched zonules → **flattened / subluxated lens**
6. **Optic nerve** – rapid **cupping**, **reversible** if early treatment
7. **Axial myopia** due to elongated globe

Intraocular Pressure

- Typical: **30–40 mmHg** (but may vary)
- Normal pediatric IOP:
    - 0–1 yr: **~11 mmHg**
    - 1–2 yr: **12 mmHg**
    - 5–12 yr: **14 mmHg**
- Measure using:
    - **Perkins**, **Tono-Pen**, **iCare**
    - Avoid **Schiotz** tonometer

Under GA

- Most anesthetics ↓IOP
- **Ketamine ↑IOP**
- Measure **immediately after airway secured**

Gonioscopy (Direct – Koeppe lens)

Findings of isolated **goniodysgenesis**:

- Smooth **hypoplastic** trabecular meshwork
- **High iris insertion**
- Fluffy tissue — **Lister's morning mist**
- Iris root vessel loops (**"Loch Ness Monster"**)

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Fundus Examination

- Avoid dilation before IOP check
- Optic disc cupping may **regress if treated early**

Differential Diagnosis

- **Red eye with epiphora:** NLDO, conjunctivitis, keratitis, uveitis, trauma
- **Corneal haze / enlargement:** Birth trauma, congenital anomalies (Peters, sclerocornea), corneal dystrophies, keratitis, metabolic disease, megalocornea
- **Optic disc cupping/anomalies:** Physiological large cup, disc pit/coloboma, optic nerve atrophy or hypoplasia

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Management

PCG causes **20% of childhood blindness** globally → **urgent** treatment needed.

General Principles

- Lifelong care with periodic EUA
- Treat associated **amblyopia**, refractive error, strabismus

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Medical Therapy (Adjunct only)

- To clear cornea while awaiting surgery or postoperative support
- **Topical/Oral CAI and β-blockers** → 25–30% IOP reduction
- **Acetazolamide**: 10–15 mg/kg/day
- **Avoid**: **Brimonidine** (risk of apnea in infants)

Surgical Therapy – Definitive

Success of angle surgeries: **80–90%** (best when done early)

1. **Goniotomy** – ab interno
    - Requires **clear cornea**
    - Incise TM ~120° under gonioscopy
    - Best if onset **3 months–1 year**
    - Can be repeated
2. **Trabeculotomy** – ab externo
    - Preferred when cornea **cloudy**
    - Limitation: difficult in **buphthalmos**, conjunctival scarring
3. **Trabeculectomy**
    - Reserved for failures of angle surgery
    - Lower success in children due to **excessive scarring**
    - Long-term risk: bleb infection → requires lifelong monitoring
4. **Glaucoma Drainage Devices (GDD)**
    - For **refractory cases**
    - Plate trimming may be needed for small eyes
    - Sometimes multiple devices required

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Cyclodestructive Procedures

- **Last resort** for refractory cases
- Reduce aqueous production (risk of hypotony/phthisis)

Prognosis

Depends on:

- **Age of onset**
- **Corneal clarity**
- **Severity at presentation**
- **Timeliness of IOP control**

Better visual outcomes if **diagnosed and treated early**.

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SECONDARY DEVELOPMENTAL GLAUCOMA

Irido-trabecular dysgenesis	Aniridia, Familial iris hypoplasia, Congenital ectropion uveae, Microcornea, Nanophthalmos	High (varies by condition)
Irido-corneal dysgenesis	Posterior embryotoxon, **Axenfeld-Rieger Syndrome**, **Peters Anomaly**	~50% in A-R & Peters

AXENFELD-RIEGER SYNDROME (A-R Syndrome)

Spectrum:

- **Axenfeld anomaly**: Posterior embryotoxon (Anteriorly displaced Schwalbe's line) + peripheral iris strands
- **Rieger anomaly**: Above + iris stromal defects
- **Rieger syndrome**: Ocular defects + **systemic developmental anomalies**

Genetics

- Autosomal dominant, high penetrance
- Genes: **PITX2**, **FOXC1**, (occasionally **PAX6**, RIEG2)
- Bilateral involvement

Ocular Features

- **Posterior embryotoxon**
- Peripheral broad **iridocorneal adhesions/strands**
- Iris stromal **hypoplasia**, **corectopia**, holes, **ectropion uveae**
- Angle abnormalities best seen on gonioscopy
- Other associations: strabismus, cataract, retinal detachment

Glaucoma

- ~**50%** cases
- Onset: infancy → early adulthood
- Severity does **not** always correlate with iris defects

Systemic Features

- **Dental anomalies**: microdontia, hypodontia
- **Facial changes**: maxillary hypoplasia, broad nasal bridge, telecanthus
- Others: redundant periumbilical skin, hypospadias, rare neurologic/cardiac defects

Management

- Lifelong monitoring
- **Start with medical therapy** (if not infantile)
- **Trabeculectomy** ± MMC preferred
- GDD or cycloablation for refractory cases/infants

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ANIRIDIA

Etiology

- **PAX6 mutation** on chromosome 11p
- Often associated with **WT1 deletion → Wilms tumor** (WAGR syndrome)

Inheritance

- **Autosomal dominant (2/3)**
- Sporadic cases: ↑ systemic risk

Clinical Features

- Bilateral **iris hypoplasia** (partial → near-total absence of iris tissue)
- ↓ Vision: **foveal hypoplasia**, **nystagmus**
- **Small cornea**, LSCD → progressive keratopathy (pannus)
- Lens: cataract, subluxation
- Gonioscopy: rudimentary iris frill

Glaucoma

- **50–75%**, typically **late childhood / adolescence**
- Mechanism: **synechial angle closure** from contracting residual iris

Systemic Concerns

- Sporadic cases: Wilms tumor surveillance per protocol

Management

- GDD preferred for glaucoma
- Trabeculectomy often fails long term
- Cycloablation if poor potential
- Supportive: lubricants, prosthetic/tinted lenses, stem-cell rescue if needed

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❖ **WAGR Syndrome**

- W – Wilms tumor
- A – Aniridia
- G – Genitourinary (GU) abnormalities
- R – Range of developmental delay *(formerly termed mental retardation)*

WAGR syndrome is associated with PAX6 and WT1 gene deletion on chromosome 11p13 and carries a high risk of Wilms tumor in children with aniridia.

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PETERS ANOMALY

- Absence of DM & Endothelium (Central Corneal Leukoma)

Pathogenesis

- Defective neural crest migration → **central corneal opacity** + posterior stromal/Descemet/endothelial defects
- Types:
  Type I – corneal only
  Type II – cornea + lens (keratolenticular touch)
  A-R + Peters overlap

Genetics: **PAX6, PITX2, FOXC1, CYP1B1**

Clinical Features

- Bilateral in >50%
- Central leukoma, shallow AC, **irido- or lenticulo-corneal adhesions**
- Systemic anomalies possible (e.g., Peters-plus syndrome)

Glaucoma

- **≈50%**, often **present at birth**
- Angle may appear deceptively normal grossly

Management

- **Trabeculotomy/trabeculectomy** if adequate AC depth
- Often requires **GDD/cycloablation**
- **Penetrating keratoplasty** frequently needed (guarded prognosis)

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GLAUCOMA IN PHACOMATOSES

Sturge–Weber Syndrome (SWS)

Encephalotrigeminal angiomatosis – congenital, usually sporadic

Key Features

- Classic triad:
  - **Port-wine stain** (V1/V2 distribution, respects midline)
  - **Ipsilateral glaucoma**
  - **Leptomeningeal angiomatosis**
- Glaucoma in **~30%**:
  - **Early-onset (<2 years: ~60%)** → trabeculodysgenesis → **buphthalmos**
  - **Late-onset** (childhood → adulthood) → **↑ episcleral venous pressure** from episcleral/ choroidal hemangiomas

Management

- **Medical therapy** may suffice in mild cases
- **Angle surgery** (goniotomy/trabeculotomy) useful in angle anomalies
- **Combined trabeculotomy–trabeculectomy**: good success in early-onset cases
  ⚠ Increased risk: **choroidal effusion / suprachoroidal hemorrhage**
- For late-onset / refractory cases:
  - **Trabeculectomy**, **GDD**, **cyclodestructive** procedures
    ⚠ Choroidal hemangiomas → risk of intraoperative **choroidal expansion**

Neurofibromatosis Type 1 (NF1)

Autosomal dominant; variable expressivity a/w Ch17 mutation

Systemic Features

- Café-au-lait spots, Lisch Nodules
- Axillary/inguinal freckling
- Sphenoid dysplasia
- Plexiform neurofibromas (often **S-shaped lids**)

Ocular Features

- Plexiform neurofibroma of upper lid → **50% risk of ipsilateral glaucoma**
- Congenital angle anomaly ± **ectropion uveae**
- Other findings: optic nerve gliomas, Lisch nodules, choroidal hamartomas

Management

- **Trabeculectomy** or **glaucoma drainage devices**
- Refractory cases may require additional surgery depending on anatomy and visual prognosis

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Iridocorneal Endothelial (ICE) Syndrome

Introduction

- Atypically unilateral condition
- Most commonly affects **middle-aged females (**20 to 50 years Female**)**
- Comprised of **three overlapping clinical variants**:
    1. **Chandler Syndrome**
    2. **Progressive (Essential) Iris Atrophy**
    3. **Iris Naevus (Cogan–Reese) Syndrome**
- **Pathophysiology**
    - Migration of abnormal endothelial cells over TM—contraction of membrane – PAS & angle closure
    - Abnormal proliferative **corneal endothelial cells** migrate across the angle and iris
    - Leads to:
        - **Angle closure** → ↑ IOP → **Glaucoma (~50%)**
        - **Corneal decompensation** due to endothelial failure
    - PCR studies show **Herpes simplex virus DNA**, suggesting viral etiology

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Clinical Diagnosis

- Overlapping features; patients may transition between subtypes
- Diagnosis based primarily on **iris and endothelial appearance**
- **Specular microscopy**: characteristic "ICE cells"
- Gonioscopy: **broad PAS**, sometimes subtle early despite high IOP

Subtypes and Key Features

I. Chandler Syndrome — *Most common*

- Cornea: **"Hammered silver/ Beaten bronze"** endothelial appearance
- Symptoms: decreased vision, **haloes** due to corneal edema
- Iris changes: minimal or mild **corectopia** (60% have no atrophy)
- Glaucoma: present but **less severe** than other forms

II. Progressive (Essential) Iris Atrophy

- Significant **iris stromal loss**
- **Corectopia**, **pseudopolycoria**
- **Severe, extensive PAS**—often anterior to Schwalbe line
- Advanced stage:
    - **Ectropion uveae**
    - **Severe secondary glaucoma**

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III. Iris Naevus (Cogan–Reese) Syndrome

- **Diffuse iris nevus** or **pigmented iris nodules**
- Iris atrophy: absent in ~50% or mild–moderate
- **Marked corectopia** may occur
- Differential: diffuse iris melanoma may mimic appearance

Management

Glaucoma

- **Medical therapy often ineffective**
- Surgical options (progressive disease)
  - **Trabeculectomy + MMC** → poor long-term success
  - **Glaucoma drainage devices (preferred)**
  - **Cyclophotocoagulation** in refractory cases

Cornea

- Early: **Hypertonic saline** for edema
- Late: **Keratoplasty** for endothelial failure

Prognosis

- **Generally poor long-term visual outcome**
- Progressive:
  - Corneal decompensation
  - Secondary glaucoma
- Lifelong follow-up required

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Malignant Glaucoma

Definition

- Malignant glaucoma, first described by Von Graefe in 1869, is a rare but serious condition characterized by a marked rise in intraocular pressure (IOP) associated with a shallow anterior chamber, usually occurring after intraocular surgery (e.g., peripheral iridectomy or trabeculectomy) in patients with primary angle-closure glaucoma (PACG).
- The condition is **not neoplastic**.
- Alternative terms based on mechanism include:
  - Ciliary block glaucoma
  - Aqueous misdirection syndrome
  - Direct lens block glaucoma
- "Aqueous misdirection syndrome" is now often preferred in literature, but this text uses **malignant glaucoma**. The international term for flat anterior chamber is **athalamia**.

Pathomechanism

The exact pathogenesis remains unclear. Postulated mechanisms include:

1. **Posterior pooling of aqueous (Aqueous misdirection syndrome)**
   - Accumulation of aqueous behind the vitreous (posterior vitreous detachment or pockets in vitreous) pushes the iris–lens or iris–vitreous diaphragm forward, leading to a shallow anterior chamber and angle-closure.

Mechanisms of posterior misdirection:

   - **Ciliary block mechanism:** Anterior rotation of ciliary body and ciliary processes pressed against the lens, along with anterior displacement of vitreous, directs aqueous posteriorly.
   - **Anterior hyaloid face block:** Intact anterior vitreous face blocks aqueous flow; breaks near vitreous base divert aqueous posteriorly.
   - **Choroidal expansion:** Increased choroidal volume pushes vitreous and iris–lens diaphragm forward, causing anterior chamber flattening and posterior aqueous accumulation.
2. **Direct lens block mechanisms**
   - Forward movement of lens–iris diaphragm due to laxity of lens zonules, which may be caused by:
     - Severe/prolonged angle-closure
     - Ciliary spasm from surgery, miotics, inflammation, or trauma

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Clinical Profile

Symptoms:

- Severe ocular pain with headache, nausea, sometimes vomiting
- Blurring of vision; occasionally marked vision loss

Signs:

- Shallow or flat anterior chamber, persistent postoperatively
- High IOP
- Negative Seidel test
- Patent iridectomy (no pupillary block)
- Absence of suprachoroidal hemorrhage
- Miotics worsen condition; cycloplegics/mydriatics may relieve it
- Myopic shift often noted due to anterior lens displacement

Clinical Forms:

1. **Classic malignant glaucoma:** After incisional surgery in PACG
2. **Aphakic/pseudophakic malignant glaucoma:** After cataract surgery (ciliovitreal or cilio-IOL block)
3. **Other forms:**
    - Miotic-induced
    - Post-laser (iridotomy, suture lysis, Nd:YAG cyclophotocoagulation)
    - Associated with retinal diseases (ROP, retinal detachment, CRVO)
    - Associated with inflammation/infection (traumatic/non-traumatic uveitis, fungal or Nocardia endophthalmitis)
    - Spontaneous cases

Diagnosis

- Based on history (recent surgery or therapy) and clinical signs
- Differentiate from:
    - Pupillary block glaucoma
    - Serous choroidal detachment
    - Suprachoroidal hemorrhage

Management

1. Medical Therapy (first-line):

- **Cycloplegics:** Atropine 1% qid to move lens–iris diaphragm backward
- **Hyperosmotic agents:** IV mannitol 2 g/kg over 45 min to reduce vitreous volume
- **Aqueous suppressants:** Topical β-blockers, α2-agonists, carbonic anhydrase inhibitors
- Avoid miotics
- Slow taper of atropine after resolution to prevent recurrence

2. Laser Therapy (second-line):

- Argon laser photocoagulation of visible ciliary processes
- Trans-scleral diode cyclophotocoagulation

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- Nd:YAG laser iridotomy or anterior hyaloidotomy (especially in aphakic/pseudophakic eyes)
- Laser posterior capsulotomy and anterior hyaloid disruption in pseudophakia

3. Surgical Therapy (if medical/laser fail):

- Needle disruption of anterior hyaloid via transcorneal approach
- Pars plana vitreous aspiration and air injection
- Pars plana vitrectomy ± lensectomy (removal of sufficient anterior vitreous gel to allow free aqueous flow) – considered the treatment of choice currently

4. Fellow Eye Management:

- Fellow eye is at risk; prophylactic laser peripheral iridotomy (PI) is recommended

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Summary:
Malignant glaucoma is a sight-threatening, postoperative complication of PACG or cataract surgery, caused by anterior rotation of the ciliary body and posterior aqueous misdirection. Early recognition, cycloplegia, aqueous suppression, hyperosmotics, laser, or surgical interventions are critical to restore anterior chamber depth and normalize IOP.

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LASER TREATMENT OF GLAUCOMA

I. Lasers for Outflow Enhancement

- **Laser Peripheral Iridotomy (LPI)** – Preferred for angle-closure glaucoma.
- **Laser Iridoplasty** – Widens angle, e.g., plateau iris.
- **Laser Pupilloplasty** – For rigid, constricted pupils.
- **Laser Sphincterotomy & Synechiotomy**
- **Goniophotocoagulation** – For neovascular glaucoma.
- **Laser Trabeculoplasty (LTP)**
  - Argon Laser Trabeculoplasty (ALT)
  - Selective Laser Trabeculoplasty (SLT)
  - Pattern Laser Trabeculoplasty (PLT)
  - Micropulse Diode Laser Trabeculoplasty (MDLT)
  - Titanium Sapphire Trabeculoplasty (TLT)
- **Excimer Laser Trabeculotomy (ELT)**
- **CO_2-assisted Deep Sclerotomy**

II. Lasers for Inflow Reduction

- Trans-scleral diode cyclophotocoagulation (TSCP)
- Endoscopic cyclophotocoagulation (ECP)- cyclocryotherapy

III. Postoperative Lasers

- Laser suturolysis
- Laser goniopuncture
- Laser reduction of dysesthetic blebs

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1. Laser Trabeculoplasty (LTP)

Purpose: Enhance aqueous outflow → **IOP reduction**

Types:

- **Argon Laser Trabeculoplasty (ALT)**
  - Mechanism: Thermal photocoagulation of trabecular meshwork → structural/physiological changes → increased outflow.
  - Indications:
    - OAG (primary/secondary) uncontrolled on topical meds
    - Noncompliant patients
    - Alternative to medical therapy
  - Expected IOP reduction: 10–40% (commonly ~25%) at 6 months
  - Complications: IOP spike, PAS, mild inflammation, cystoid macular edema
- **Selective Laser Trabeculoplasty (SLT)**
  - Mechanism: Selective photothermolysis of pigmented TM cells → minimal thermal damage, stimulates macrophage recruitment & extracellular matrix remodeling.

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- Indications: Same as ALT + patients with failed ALT
- Expected IOP reduction: 10–40%, ~25% common at 6 months
- Advantages: Repeatable, less tissue damage
- Complications: Transient IOP spike, mild iritis

- **Pattern Laser Trabeculoplasty (PLT):** Multi-spot, computer-guided laser for OAG; IOP reduction ≈ SLT.
- **Micropulse Diode Laser Trabeculoplasty (MDLT):** Sub-threshold diode pulses stimulate TM → IOP ↓; minimal complications.
- **Titanium Sapphire Trabeculoplasty (TLT):** Near-infrared laser, deep penetration, repeatable; OAG/OHT.
- **Excimer Laser Trabeculotomy (ELT):** Excimer laser creates TM microperforations → immediate IOP drop; risk of hemorrhage.
- **Carbon Dioxide Laser-Assisted Deep Sclerectomy (CO_2 DL):** Laser deroofs Schlemm's canal → improved outflow; may cause scarring/astigmatism.

2. Laser Peripheral Iridotomy (LPI)

- Mechanism: Creates a hole in the peripheral iris → bypass pupillary block → **IOP reduction** in angle-closure glaucoma
- Pre-treatment: Pilocarpine 2% – 1 drop every 5 minutes × 5 doses
- Setting:
  - Energy: 3–10 mJ
  - Pulses: 1–3 pulses/shot
  - PI size: 300–500 µm
- Indications (Absolute):
  - Acute angle-closure glaucoma
  - Primary angle-closure glaucoma
  - Pupillary block (aphakic/pseudophakic)
- Relative: Narrow angles, travel constraints, positive provocative test
- Complications: Transient IOP rise, iritis, iris bleeding, focal cataract, dysphotopsia

Laser Peripheral Iridoplasty (LPIp): Contraction burns pull peripheral iris → open angle; for plateau iris/residual closure; complications: IOP spike, mild iritis, synechiae.

Laser Pupilloplasty: Burns near pupil dilate rigid pupil → improve outflow; for pupillary block, aphakia/pseudophakia; moderate IOP reduction.

Laser Sphincterotomy: Argon laser transects iris sphincter → better aqueous flow; for pupillary block; moderate IOP reduction.

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Goniophotocoagulation: Laser ablates angle vessels → prevent neovascular blockage; for neovascular glaucoma adjunct to PRP; moderate IOP reduction.

II. Lasers for Inflow Reduction

1. Trans-scleral Diode Cyclophotocoagulation (TSCPC)

- Mechanism: Coagulates pigmented ciliary epithelium → **reduced aqueous secretion → IOP reduction**
- Indications:
  - Eyes with poor visual potential
  - Failed filtering surgery or high surgical risk
  - Secondary glaucoma (neovascular, post-keratoplasty)
- Complications: Temporary IOP rise, inflammation, hypotony, phthisis, rare suprachoroidal hemorrhage

2. Endoscopic Cyclophotocoagulation (ECP)

- Mechanism: Direct ablation of ciliary processes via endoscope → **reduced aqueous secretion**
- Indications: Similar to TSCPC; can be used in eyes with useful vision
- Complications: Mild pain, temporary IOP rise, 10% risk of uveitis/hemorrhage

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Signs of Patent Peripheral Iridotomy (PI)

- Sudden gush of aqueous
- Retroillumination through PI (patent PI)
- Deepening of anterior chamber (AC)
- Release of iris pigment
- Visualization of the anterior lens capsule

Complications of Peripheral Iridotomy (PI)

- Corneal burn
- Uveitis
- ↑ IOP spike
- Hyphema
- Lens injury
- Corectopia
- Diplopia
- Glare
- Malignant glaucoma

Laser Suture Lysis (LSL)

- Timing: 3 days–2 months after trabeculectomy
- Settings: Argon laser, 50 µm spot size, 200 mW power, 0.02 s duration
- Procedure: Laser lysis of scleral flap sutures
- Indication: High IOP due to an overly tight scleral flap after trabeculectomy

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SURGICAL MANAGEMENT OF GLAUCOMA

1. INTRODUCTION

Glaucoma surgery is indicated when target intraocular pressure (IOP) cannot be achieved or maintained with maximal tolerated medical therapy and/or laser procedures. The ultimate goal is **preservation of visual function** by preventing further optic nerve damage through sustained IOP reduction.

Principles of surgical management:

- Individualized target IOP based on stage of disease
- Long-term efficacy with minimal complications
- Preservation of conjunctiva for future surgery

Surgical methods reduce IOP by:

1. **Enhancing aqueous outflow** (trabecular, uveoscleral, or subconjunctival)
2. **Reducing aqueous production** (ciliary body ablation)

CLASSIFICATION OF GLAUCOMA SURGERIES

A. Based on Type of Glaucoma

1. Congenital / Developmental Glaucoma

- Primary: Goniotomy, trabeculotomy
- Failed cases: Combined trabeculotomy-trabeculectomy
- Refractory: Glaucoma drainage devices

2. Primary Open-Angle Glaucoma (POAG)

- Trabeculectomy ± antimetabolites
- MIGS (mild–moderate disease)
- Drainage devices (failed trab)

3. Primary Angle-Closure Glaucoma (PACG)

- Laser / surgical PI
- Lens extraction
- Trabeculectomy or combined surgery

4. Secondary Glaucoma

- Treat underlying cause
- Goniosynechiolysis, vitrectomy, lens extraction

5. Intractable / Absolute Glaucoma

- Drainage devices
- Cyclodestructive procedures

B. Based on Mechanism of IOP Reduction

I. Aqueous Outflow Enhancement

- External filtration: Trabeculectomy
- Non-penetrating: Deep sclerectomy, viscocanalostomy

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- Internal (angle-based): Goniotomy, trabeculotomy, GATT
- Canal-based: Canaloplasty, Trabectome, iStent
- Subconjunctival shunts: Ahmed, Baerveldt

II. Aqueous Inflow Reduction

- Cyclocryotherapy
- Trans-scleral CPC
- Endoscopic CPC

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5. SURGICAL PERIPHERAL IRIDECTOMY

Indications

- Acute & chronic angle closure
- Phacomorphic glaucoma
- Iris bombe
- Prophylaxis in PAC

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6. FILTRATION SURGERIES

6.1 Trabeculectomy (Gold Standard)

Definition: Guarded filtration surgery creating a fistula between AC and subconjunctival space.

Mechanism: Bypasses trabecular meshwork → subconjunctival drainage via filtering bleb.

Indications for Glaucoma Surgery

- POAG: uncontrolled on meds/laser
- PACG: PAS >50%, pupillary block
- Congenital/developmental glaucoma: failed goniotomy/trabeculotomy
- Secondary glaucoma: refractory to therapy
- Progressive optic nerve/field damage
- Poor compliance or ocular tissue limiting meds
- Advanced glaucoma + cataract → combined surgery

Relative Contraindications

- Blind, painful eye
- Rubeosis iridis
- Active iritis
- Conjunctival scarring / thin sclera
- Failed prior filtration with antimetabolites
- Congenital, aphakic, uveitic glaucoma

Poor Prognosis in Filtering Surgery

- Young age (<40), Black race
- Active inflammation / uveitis, Traumatic Glaucoma
- Neovascular glaucoma, Prev. failed trab
- Aphakia / Pseudophakia
- Congenital glaucoma

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- Cicatrizing disorders (SJS, ocular pemphigoid)
- Prior ocular surgery (PK, scleral buckle, previous trab)
- Chronic topical medications

Preoperative Evaluation

- Control IOP pre-op
- Assess conjunctival integrity
- Gonioscopy for PAS
- Treat NVI with PRP/anti-VEGF

Surgical Steps

1. Anesthesia and exposure
2. Conjunctival flap (fornix/limbal based)
3. Partial thickness scleral flap
4. Antimetabolite application (MMC 0.02–0.04%)
5. Paracentesis
6. Sclerostomy
7. Peripheral iridectomy
8. Scleral flap suturing (fixed/releasable)
9. Watertight conjunctival closure

Antimetabolites

Indications for Antimetabolites: Previous failed filtration, aphakic glaucoma, inflammatory/post-trauma/neovascular/ICE glaucoma, long-term topical meds (>3 yrs), cicatrizing conjunctivitis.

Mitomycin C (MMC):

- **MOA:** Alkylating agent → binds DNA, inhibits DNA-dependent RNA synthesis, suppresses fibroblast proliferation.
- **Side effects:** Hypotony, thin bleb, leaks, scleral thinning, delayed healing, rare endophthalmitis, punctal stenosis.

5-Fluorouracil (5FU):

- **MOA:** Pyrimidine analog; S-phase specific, folic acid antagonist → inhibits DNA/RNA synthesis, fibroblast proliferation → enhances bleb function.
- **Side effects:** Conjunctival irritation, epithelial defects, corneal toxicity, delayed wound healing, rare infection.

Bleb Evaluation

Ideal Bleb: Posterior, diffuse, moderately elevated, avascular, epithelial microcysts

Bleb Grading Systems

1. Moorfields Bleb Grading System (MBGS)

- Central area
- Maximal area

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- Height
- Vascularity

2. Indiana Bleb Appearance Grading Scale (IBAGS) – HEEVS

- H – Height
- E – Extent
- E – Edge
- V – Vascularity
- S – Seidel test

Signs of a Failing Bleb

- ↓ Bleb height
- ↓ Bleb wall thickness
- ↑ Vascularity
- Loss of transparency
- Loss of microcysts
- ↑ Intraocular pressure (IOP)

Postoperative Eye Evaluation:

- Bleb: height, extent, microcysts
- Aqueous leak: present/absent
- Scleral flap sutures visibility
- IOP
- Cornea clarity
- Anterior chamber: depth, inflammation
- Hyphema
- Choroidal detachment / suprachoroidal hemorrhage
- Optic disc and macula appearance

Complications:

A. Intraoperative

- **Conjunctival buttonhole**
  - *Cause:* Tear during flap creation/closure
  - *Prevention:* Gentle handling, avoid toothed forceps
  - *Management:* Small → figure-of-8/mattress suture; Large → running suture or shift site
- **Scleral flap tear / disinsertion**
  - *Management:* Small → 10-0 nylon; Complete → rotate sclera / patch graft
- **Bleeding**
  - *Risk:* Long-term topical drugs
  - *Management:* Irrigation, cautery, pressure, AC air bubble
- **Suprachoroidal hemorrhage (SCH)**
  - *Risk:* High IOP, aphakia, vitrectomy, anticoagulants
  - *Features:* Sudden pain, vision loss, shallow AC

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  - *Prevention:* Lower IOP, control BP, slow decompression
  - *Management:* Close wound, reform AC, IV mannitol, posterior sclerotomy if needed
- **Choroidal effusion**
  - *Cause:* Hypotony, venous congestion
  - *Management:* Cycloplegics + topical steroids; drainage if AC flat
- **Others:** Vitreous loss, lens injury, Descemet detachment

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B. Early Postoperative

- **Hypotony / shallow AC:** Due to leak, overfiltration, choroidal detachment → observe or reform AC
- **Conjunctival leak:** Patch, bandage CL, glue, surgical repair
- **Excessive filtration:** Reduce steroids, AC reformation, surgical revision if persistent
- **Hypotony / deep AC:** Acceptable early; persistent → hypotony maculopathy → bleb compression, blood injection
- **Elevated IOP / flat AC:** Malignant glaucoma, pupillary block, delayed SCH → cycloplegics, aqueous suppressants, PPV if needed
- **Elevated IOP / deep AC:** Tight flap or blockage → laser suture lysis, surgical revision
- **Failing bleb:** Flat, vascular → increase steroids, massage, suture lysis, 5-FU, revision
- **Encapsulated bleb (Tenon's cyst):** Needling + antimetabolite, surgery if refractory
- **Others:** Hyphema, uveitis, dellen, snuff-out, decompression retinopathy

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C. Late Postoperative

- **Late bleb failure:** Scleral/conjunctival fibrosis → laser or surgical revision, GDD
- **Leaking bleb:** Thin/avascular, antimetabolite-related → medical therapy, patch/cl, surgery
- **Bleb-related infections:**
  - Blebitis → topical ± oral antibiotics
  - Endophthalmitis → intravitreal + systemic antibiotics
- **Others:** Cataract, overhanging bleb, corneal decompensation, ptosis, sympathetic ophthalmia

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7. NON-PENETRATING GLAUCOMA SURGERY (NPGS)

Types

- Deep sclerectomy
- Viscocanalostomy
- Sinusotomy

Advantages: Reduced hypotony, no AC entry

Disadvantages: Less IOP reduction, technically demanding

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8. GONIOTOMY & TRABECULOTOMY

Goniotomy

- Indication: Congenital glaucoma with clear cornea
- Incision of trabecular meshwork under gonioscopy

Trabeculotomy

- Indication: Hazy cornea, failed goniotomy
- Can be conventional or 360° microcatheter-assisted

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9. GATT (Gonioscopy-Assisted Transluminal Trabeculotomy)

Indications: POAG, juvenile glaucoma
Advantages: Conjunctiva-sparing, 360° TM treatment

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10. COMBINED CATARACT + GLAUCOMA SURGERY

Indications: Visually significant cataract with glaucoma requiring surgery

- 2 meds or intolerance + cataract
- Non-compliant/low socioeconomic
- One-eyed with moderate glaucoma
- Moderate glaucoma to prevent post-op IOP spike
- High cataract risk post-trabeculectomy

Types: Phacotrabeculectomy (single-site / two-site)
Limitation: Less IOP reduction than trabeculectomy alone

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11. MINIMALLY INVASIVE GLAUCOMA SURGERY (MIGS)

Indications: Mild–moderate glaucoma
Types:

- Trabecular: Trabectome, Kahook Dual Blade
- Bypass: iStent, Hydrus
- Subconjunctival: XEN, MicroShunt

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12. GLAUCOMA DRAINAGE DEVICES

Indications: Refractory glaucoma, failed trabeculectomy, neovascular glaucoma
Valved: Ahmed **Non-valved:** Baerveldt, Molteno
Complications:

- **Early:** Hypotony, AC shallowing, choroidal effusion, tube occlusion
- **Late:** Tube exposure, corneal decompensation, diplopia, endophthalmitis
- *Prevention:* Correct tube placement, patch graft, posterior plate placement

---

13. CYCLODESTRUCTIVE PROCEDURES

Indications: Absolute or refractory glaucoma
Types:

- Cyclocryotherapy, Diode CPC, Endoscopic CPC
- **Complications:** Hypotony, vision loss, uveitis, pain, phthisis, RD, endophthalmitis

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RECENT ADVANCES IN GLAUCOMA

1. RECENT ADVANCES IN MEDICAL THERAPY

1.1 Chronology of Antiglaucoma Medications (Key Milestones)

- **1875** – Pilocarpine
- **1978** – Timolol
- **1996** – Latanoprost, Brimonidine
- **2001** – Bimatoprost, Travoprost
- **2017** – **Latanoprostene Bunod (Vyzulta)**, **Netarsudil (Rhopressa)**
- **2020** – **Durysta (Bimatoprost intracameral implant)**

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2. NEWER PHARMACOLOGICAL AGENTS

2.1 Rho-Kinase (ROCK) Inhibitors

Examples

- Ripasudil 0.4%
- Netarsudil 0.02%

Mechanism

- ROCK pathway regulates trabecular meshwork (TM) tone
- Inhibition → ↓ actin stress fibers → TM relaxation → ↑ trabecular outflow

Additional Benefits

- Neuroprotection
- Increased ocular blood flow
- Preserves corneal endothelium
- Enhances axonal regeneration

Clinical Importance

- Effective during both **diurnal and nocturnal periods**

2.2 Nitric Oxide (NO) Donors

Example

- **Latanoprostene Bunod 0.024%**

Mechanism

- Dual action:
  - NO → relaxes TM & Schlemm's canal → ↑ trabecular outflow
  - PG analog → ↑ uveoscleral outflow

Evidence

- **LUNAR & APOLLO studies**: Non-inferior to Timolol
- **VOYAGER study**: Superior IOP reduction compared to latanoprost alone

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2.3 New Prostaglandin Pathway Targets

- Current PGAs act mainly on **FP receptors**
- Future targets:
  - **PGE2 agonists** – Sulprostone, Butaprost

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- **PGI2 analogue** – Iloprost
- **EP receptor agonists** – DE-117 (Phase II)
- **ONO-9054** – Dual FP & EP3 agonist

2.4 Adenosine A1 Receptor Agonist

Drug

- **Trabodenoson (INO-8875)**

Mechanism

- A1 receptor activation → ↓ aqueous production + ↓ outflow resistance

Status

- Phase III trials
- Well tolerated with minimal systemic side effects

2.5 siRNA Therapy

Drug

- **Bamosiran (SYL-040012)**

Mechanism

- Gene silencing of β-2 adrenergic receptors in ciliary body
- Avoids systemic β-blocker side effects

2.6 Cannabinoids

Mechanism

- CB1 & CB2 receptor activation → ↑ outflow facility

Limitations

- Short duration
- Significant systemic and ocular side effects
- Not routinely recommended

2.7 Ocular Hypotensive Lipids (OHL)

Examples

- AGN-191129, AGN-192024

Features

- Twice daily dosing
- Sustained IOP reduction
- Favorable safety profile

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3. NEUROPROTECTION IN GLAUCOMA

3.1 NMDA Receptor Antagonists

Drug

- **Memantine**

Mechanism

- Blocks glutamate-mediated excitotoxicity

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- Prevents RGC apoptosis
- Non-IOP-lowering neuroprotective strategy

3.2 Calcium Channel Blockers

Examples

- Nimodipine, Verapamil

Benefits

- Improved optic nerve blood flow
- Useful in **normal tension glaucoma**
- Enhances bleb survival post-filtering surgery

3.3 Other Neuroprotective Strategies

- Alpha-2 agonists (Brimonidine)
- Neurotrophic factors (BDNF, CNTF, NGF)
- Antioxidants (Resveratrol, Alpha-lipoic acid)
- Apoptosis inhibitors

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4. NOVEL DRUG DELIVERY SYSTEMS

4.1 Intracameral Implants

Durysta (Bimatoprost)

- FDA approved (2020)
- Biodegradable implant
- Sustained release for 3–4 months

iDose Travoprost

- Titanium implant anchored in TM
- Sustained release >6 months
- Phase III trials ongoing

4.2 Other Delivery Platforms

- Bimatoprost topical ring
- Travoprost punctal plugs
- Contact lens-based drug delivery

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5. SURGICAL ADVANCES IN GLAUCOMA

5.1 Micro-Invasive Glaucoma Surgery (MIGS)

Key Characteristics

- Ab-interno
- High safety profile
- Minimal tissue disruption
- Faster recovery

5.2 MIGS Targets & Devices

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A. Trabecular Outflow

- iStent G1, G2 Inject
- Hydrus Microstent
- KDB Goniotomy
- GATT
- OMNI Surgical System

Hydrus (HORIZON Trial)

- Superior IOP control vs phaco alone
- Reduced medication dependence
- Mild–moderate POAG

B. Subconjunctival Space

- XEN Gel Stent
- Preserflo Microshunt
- Ex-Press shunt

C. Suprachoroidal Space

- SOLX Gold Microshunt
- CyPass (withdrawn)
- iStent Supra

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6. CYCLOABLATIVE PROCEDURES

Modern Techniques

- **Micropulse Transscleral CPC**
- **Endoscopic Cyclophotocoagulation (ECP)**
- **High-Intensity Focused Ultrasound (HIFU)**

Micropulse CPC Advantages

- Reduced collateral damage
- Suitable for eyes with visual potential
- Better safety than continuous-wave CPC

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7. LASER ADVANCES

Micropulse Laser Trabeculoplasty (MLT)

- Less inflammation than ALT/SLT
- Repeatable
- Comparable efficacy

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8. GLAUCOMA DRAINAGE DEVICES (GDDs)

Newer Devices

- **Paul Glaucoma Implant**
- **Aurolab Aqueous Drainage Implant (AADI)**

Advantages

- Larger end-plate area
- Better long-term IOP control
- Cost-effective (AADI)

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9. PREVENTION OF POST-SURGICAL FIBROSIS

- Anti-VEGF therapy
- TGF-β inhibitors
- PRM-151
- Gene therapy (p53 transfer)

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10. ARTIFICIAL INTELLIGENCE IN GLAUCOMA

Applications

- AI-based fundus & OCT screening
- Visual field progression prediction
- Risk stratification
- Tele-glaucoma (low-resource settings)
- Clinical decision support systems

EXAM & VIVA PEARLS

- Latanoprostene Bunod = Dual mechanism PGA
- ROCK inhibitors act at trabecular cytoskeleton
- Durysta = first FDA-approved intracameral PGA
- Hydrus > iStent (2 stents) for success rate
- Micropulse CPC safer than continuous wave

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Landmark Glaucoma Trials

1. EAGLE Study (Effectiveness in Angle-closure Glaucoma of Lens Extraction)

- Compared: Clear Lens Extraction (CLE) vs LPI + medications
- Inclusion: PAC (IOP >30 mmHg) / PACG, Age >50 years, No cataract
- Conclusion: Early CLE is superior → Better IOP control, fewer medications, better quality of life, cost-effective.

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2. ZAP Study (Zhongshan Angle-Closure Prevention Trial)

- Compared: Prophylactic LPI vs Observation (PACS)
- Conclusion: LPI reduces progression, but absolute risk is very low → Routine prophylactic LPI is not recommended for all PACS.

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3. LiGHT Trial (Laser in Glaucoma and Ocular Hypertension Trial)

- Compared: SLT vs Eye drops
- Population: POAG, OHT
- Conclusion: SLT is first-line therapy → Similar/better IOP reduction, drop-free control, cost-effective, fewer surgeries.

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4. TVT Study (Tube Versus Trabeculectomy Study)

- Compared: Tube shunt (Baerveldt) vs Trabeculectomy + MMC
- Indications: Failed trabeculectomy, previous cataract surgery, uncontrolled glaucoma
- Conclusion: Tube shunts superior in previously operated eyes → Higher success, lower failure, fewer complications.

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5. Bhaktapur Glaucoma Study (2013, Nepal)

- Type: Population-based, Age ≥40 years
- Prevalence: Glaucoma 2.9%, POAG 1.24%, PACG 0.34%, Secondary glaucoma 0.73%
- Key findings: Mean IOP 12.3 mmHg, Mean VCDR 0.26, Upper normal VCDR 0.6
- Conclusion: POAG most common; PACG causes greater visual morbidity and blindness.

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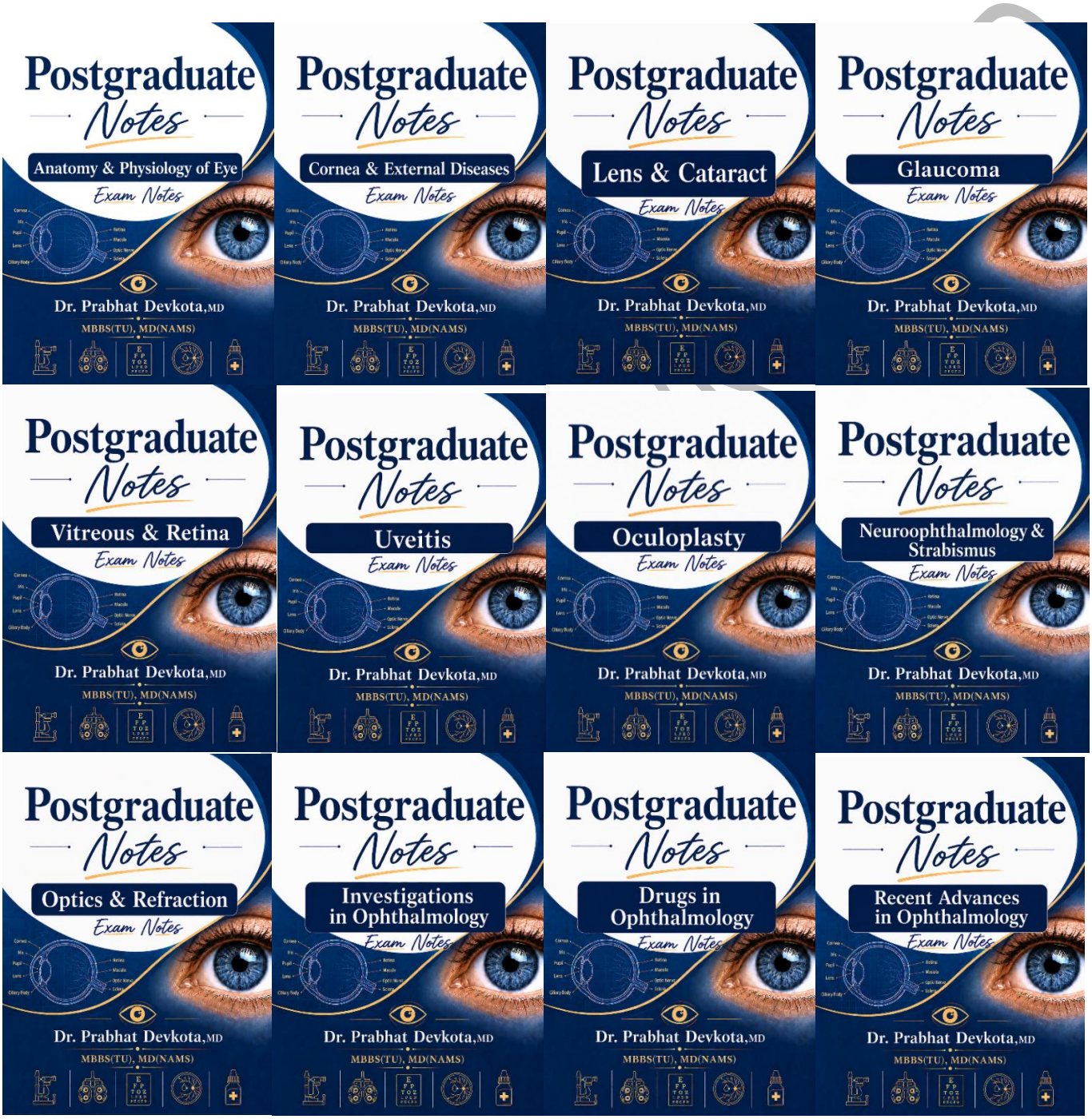

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